



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

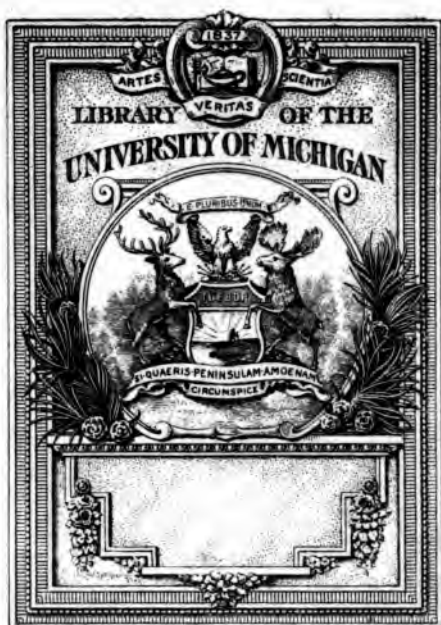
Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

A

3 9015 00382 628 9

University of Michigan - BUHR





610.5
R89
M5t

MEDICO-CHIRURGICAL
TRANSACTIONS.
75940

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON.

VOLUME THE SIXTY-FIRST.

LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

1878.

MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON.

SECOND SERIES.
VOLUME THE FORTY-THIRD.



LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

1878.

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.

**ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.**

PATRON.
THE QUEEN.

**OFFICERS AND COUNCIL,
ELECTED MARCH 1, 1878.**

President.

CHARLES WEST, M.D

VICE-PRESIDENTS.	{	WILLIAM WOOD, M.D.
		RICHARD QUAIN, M.D., F.R.S.
		JOHN COOPER FORSTER.
		JOHN WOOD, F.R.S.
TREASURERS.	{	WILLIAM WEGG, M.D.
		JOHN BIRKETT.
SECRETARIES.	{	JAMES ANDREW, M.D.
		TIMOTHY HOLMES.
LIBRARIANS.	{	GEORGE JOHNSON, M.D., F.R.S.
		WILLIAM SCOVELL SAVORY, F.R.S.
OTHER MEMBERS OF COUNCIL.	{	ROBERT BARNES, M.D.
		WILLIAM CHAPMAN BEGLEY, M.D.
		CHARLES HANDFIELD JONES, M.B., F.R.S.
		OCTAVIUS STURGES, M.D.
		E. SYMES THOMPSON, M.D.
		WILLIAM MORRANT BAKER.
		HENRY HOWARD HAYWARD.
		MATTHEW BERKELEY HILL.
	{	ARTHUR B. R. MYERS.
		ROBERT JOHN SPITTA, M.D.

THE ABOVE FORM THE COUNCIL.

**RESIDENT ASSISTANT-LIBRARIAN.
BENJAMIN ROBERT WHEATLEY.**

A LIST OF THE PRESIDENTS OF THE SOCIETY FROM ITS FORMATION.

ELECTED

- 1805. WILLIAM SAUNDERS, M.D.
- 1808. MATTHEW BAILLIE, M.D.
- 1810. SIR HENRY HALFORD, BART., M.D., G.C.H.
- 1813. SIR GILBERT BLANE, BART., M.D.
- 1815. HENRY CLINE.
- 1817. WILLIAM BABINGTON, M.D.
- 1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
- 1821. JOHN COOKE, M.D.
- 1823. JOHN ABERNETHY.
- 1825. GEORGE BIRKBECK, M.D.
- 1827. BENJAMIN TRAVERS.
- 1829. PETER MARK ROGET, M.D.
- 1831. SIR WILLIAM LAWRENCE, BART.
- 1833. JOHN ELLIOTSON, M.D.
- 1835. HENRY EARLE.
- 1837. RICHARD BRIGHT, M.D., D.C.L.
- 1839. SIR BENJAMIN COLLINS BRODIE, BART., D.C.L.
- 1841. ROBERT WILLIAMS, M.D.
- 1843. EDWARD STANLEY.
- 1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
- 1847. JAMES MONCRIEFF ARNOTT.
- 1849. THOMAS ADDISON, M.D.
- 1851. JOSEPH HODGSON.
- 1853. JAMES COPLAND, M.D.
- 1855. CÆSAR HENRY HAWKINS.
- 1857. SIR CHARLES LOCOCK, BART., M.D.
- 1859. FREDERIC CARPENTER SKEY.
- 1861. BENJAMIN GUY BABINGTON, M.D.
- 1863. RICHARD PARTRIDGE.
- 1865. SIR JAMES ALDERSON, M.D., D.C.L.;
- 1867. SAMUEL SOLLY.
- 1869. SIR GEORGE BURROWS, BART., M.D., D.C.L.
- 1871. THOMAS BLIZARD CURLING.
- 1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
- 1875. SIR JAMES PAGET, BART., D.C.L., LL.D.
- 1877. CHARLES WEST, M.D.

FELLOWS OF THE SOCIETY APPOINTED BY
THE COUNCIL AS REFEREES OF PAPERS,
FOR THE SESSION OF 1878-79.

BARCLAY, ANDREW WHYTE, M.D.
BURROWS, SIR GEORGE, BART., M.D., D.C.L., F.R.S.
CALLENDER GEORGE WILLIAM, F.R.S.
CHOLMELEY, WILLIAM, M.D.
CHURCH, WILLIAM SELBY, M.D.
CLARK, FREDERICK LE GROS, F.R.S.
CURLING, THOMAS BLIZARD, F.R.S.
DAVIS, JOHN HALL, M.D.
FAGGE, CHARLES HILTON, M.D.
HABERSHON, SAMUEL OSBORNE, M.D.
HAWKINS, CÆSAR HENRY, F.R.S.
HEWETT, PRESCOTT GARDNER, F.R.S.
HEWITT, GRAILY, M.D.
HUTCHINSON, JONATHAN.
LANE, JAMES ROBERT.
LEE, HENRY.
MARSHALL, JOHN, F.R.S.
PAVY, FREDERICK WILLIAM, M.D., F.R.S.
POLLOCK, GEORGE DAVID.
POLLOCK, JAMES E., M.D.
POWER, HENRY.
PRIESTLEY, WILLIAM OVEREND, M.D.
RADCLIFFE, CHARLES BLAND, M.D.
REES, GEORGE OWEN, M.D., F.R.S.
RINGER, SYDNEY, M.D.
SANDERSON, J. BURDON, M.D., F.R.S.
SIEVEKING, EDWARD HENRY, M.D.
SIMON, JOHN, C.B., D.C.L., F.R.S.
SMITH, SPENCER.
SOUTHEY, REGINALD, M.D.
THOMPSON, REGINALD, M.D.
TILT, EDWARD JOHN, M.D.
WEBER, HERMANN, M.D.
WILKS, SAMUEL, M.D., F.R.S.
WILLIAMS, JOHN, M.D.

TRUSTEES OF THE SOCIETY.

SIR GEORGE BURROWS, BART., M.D., D.C.L., F.R.S.
CÆSAR HENRY HAWKINS, F.R.S.
THOMAS BLIZARD CURLING, F.R.S.

TRUSTEES OF THE MARSHALL HALL MEMORIAL FUND.

WALTER BUTLER CHEADLE, M.D.
WILLIAM OGLE, M.D.
THOMAS SMITH.

LIBRARY COMMITTEE FOR THE SESSION OF 1878-79.

CHARLES HILTON FAGGE, M.D.
J. WICKHAM LEGG, M.D.
JOSEPH FRANK PAYNE, M.B.
J. BURDON SANDERSON, M.D., F.R.S.
JOHN WILLIAMS, M.D.
JOHN CROFT.
ROBERT FARQUHARSON, M.D.
JOHN WHITAKER HULKE, F.R.S.
HENRY POWER.
S. JAMES A. SALTER, F.R.S.
Hon. Secs. { JAMES ANDREW, M.D.
 { TIMOTHY HOLMES.
Hon. Libs. { GEORGE JOHNSON, M.D., F.R.S.
 { WILLIAM S. SAVORY, F.R.S.

FELLOWS
OF THE
ROYAL MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

P.—President.	V.P.—Vice-President.
T.—Treasurer.	S.—Secretary.
L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1878.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

- 1846 ***ABERCROMBIE, JOHN, M.D.**, Physician to the Cheltenham General Hospital ; 13, Suffolk square, Cheltenham.
- 1877 **ABERCROMBIE, JOHN, M.B.**, St. Bartholomew's Hospital.
- 1851 ***ACLAND, HENRY WENTWORTH, M.D., F.R.S.**, Honorary Physician to H.R.H. the Prince of Wales ; Physician to the Radcliffe Infirmary ; Regius Professor of Medicine, and Clinical Professor in the University of Oxford:
- 1847 **ACOSTA, ELISHA, M.D.**, 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

- 1852 ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.
- 1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.
- 1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.
- 1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.
- 1866 ALBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.
- 1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 36, Bryanston street, Portman square. *Trans.* 2.
- 1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.
- 1862 ANDREW, JAMES, M.D., *Secretary*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878. *Trans.* 1.
- 1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.
- 1870 ARNOTT, Rev. HENRY, Braeside, Beckenham.
- 1819 †ARNOTT, JAMES MONCIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.
- 1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly. C. 1821, 1830-31.
1874. AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

Elected

- 1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital ; 7, Camden crescent, Dover, Kent.
- 1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital ; 20A, Temple row, Birmingham.
- 1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary ; 102, Friargate, Derby.
- 1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital ; Surgeon to the Evelina Hospital for Sick Children ; 26, Wimpole street, Cavendish square. C. 1878. *Trans.* 5.
- 1869 BAKEWELL, ROBERT HALL, M.D., Dunedin, New Zealand.
- 1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General ; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.
- 1848 BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board ; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Trans.* 5.
- 1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals ; Consulting Physician to the Coombe Hospital ; 10, Merrion square east, Dublin.
- 1847 †BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital ; Medical Officer of Health for Chelsea ; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.
- 1862 BARKER, EDGAR, 21, Hyde park street.
- 1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital ; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.
- 1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to Charing Cross Hospital, and to the Hospital for Sick Children ; 10, Montague street, Russell square.

Elected

- 1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Trans.* 4.
- 1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Baywater.
- 1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.
- 1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. *Trans.* 5.
- 1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.
- 1874 BAXTER, EVAN BUCHANAN, M.D., Professor of Materia Medica at King's College, London; Assistant Physician to King's College Hospital; 28, Weymouth street, Portland place.
- 1875 BEACH, FLETCHER, M.B., Medical Superintendent, Clapton Idiot Asylum, Lower Clapton.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Trans.* 1.
- 1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.
- 1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.
- 1871 BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.
- 1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877-8.
- 1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

Elected

- 1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Professor of Anatomy in the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square.
- 1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.
- 1877 BENNETT, WILLIAM HENRY, Surgeon to the Belgrave Hospital for Children; 22, James Street, Buckingham Gate.
- 1845 †BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.
- 1820 BERTIN, STEPHEN, Paris.
- 1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.
- 1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.
- 1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.
- 1878 BINDON, WILLIAM JOHN VEREKER, M.D., 2, Elm Villas, Kilburn.
- 1854 BIRD, PETER HINCKES, F.L.S., 1, Norfolk square, Sussex gardens, Hyde park.
- 1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- 1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.
- 1851 †BIRKETT, JOHN, F.L.S., *Treasurer*, Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. *Trans.* 8. *Sci. Com.*
- 1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

Elected

- 1843 †BLACK, PATRICK, M.D., Consulting Physician to St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.
- 1840 BLAKISTON, PEYTON, M.D., F.R.S.
- 1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.
- 1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.
- 1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.
- 1823 BOJANUS, LOUIS HENRY, M.D., Wilna.
- 1846 †BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.*
- 1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]
- 1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.
- 1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.
- 1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.
- 1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]
- 1867 *BRETT, ALFRED T., M.D., Watford, Herts.
- 1876 BRIDGES, ROBERT, M.B., Casualty Physician to St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children; and Physician to the Great Northern Hospital; 52, Bedford square.

Elected

- 1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.
- 1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 3.
- 1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.
- 1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.
- 1844 †BROOKE, CHARLES, M.A., F.R.S., Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72. V.P. 1875-76.
- 1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.*
- 1878 BROWNE, JAMES CRICHTON, M.D., 7, Cumberland Terrace, Regent's Park.
- 1874 BRUCE, JOHN MITCHELL, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 60, Queen Anne street.
- 1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica at the University of London; 50, Welbeck street, Cavendish square.
- 1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Trans.* 8; *Pro.* 1. *Sci. Com.*
- 1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.
- 1823 BUCHANAN, B. BARTLET, M.D.

Elected

- 1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.
- 1864 BUCKLE, FLEETWOOD, M.D.
- 1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; 39, Wimpole street, Cavendish Square.
- 1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.
- 1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Trans.* 2.
- 1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.
- 1873 BUTLIN, HENRY TRENTAM, Surgical Registrar to St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.
- 1871 BUTT, WILLIAM F., 25, Park street, Park lane.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.
- 1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.
- 1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to the Charter House; Examiner in Anatomy at the University of London; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.*
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place.

Elected

- 1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 69, Wimpole street, Cavendish square, W. *Trans.* 1.
- 1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*
- 1868 CAVAFY, JOHN, M.D., Senior Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; 2, Upper Berkeley street, Portman square.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 58, Welbeck street, Cavendish square.
- 1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.
- 1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; Shrubs Hill House, Sunningdale, Middlesex. C. 1861. V.P. 1867. L. 1869-72. *Trans.* 1.
- 1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.
- 1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.
- 1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 10, Mitre court Chambers, Temple.
- 1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.
- 1873 *CHISHOLM, EDWIN, M.D., Camden, near Sydney, New South Wales.

Elected

- 1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.
- 1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.
- 1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square.
- 1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.
- 1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 63, Warrior square, St. Leonards-on-Sea. S. 1847-9. V.P. 1855-6. *Trans.* 5.
- 1848 CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.
- 1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.
- 1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.
- 1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.
- 1857 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.
- 1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 4, Suffolk place, Pall Mall East. *Trans.* 2.
- 1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.
- COLLEY, DAVIES, see *Davies-Colley*.
- 1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.
- 1865 COOPER, ALFRED, Surgeon to the Lock Hospital; Assistant Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

Elected

- 1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.-P. 1873-4.
- 1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.
- 1860 *CORY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.
- 1853 CORY, WILLIAM GILLETT, M.D.
- 1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.
- 1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876.
- 1877 COUPLAND, SIDNEY, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 7, Nottingham place.
- 1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.
- 1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D. C. 1853-4.
- 1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.
- 1873 CREIGHTON, CHARLES, M.B., Demonstrator of Anatomy, University of Cambridge, Anatomical Museum, Cambridge.
- 1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.
- 1874 CRIPPS, WILLIAM HARRISON, Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital; 61, Pall Mall. *Trans.* 1.

Elected

- 1847 †CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Middlesex Hospital; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.
- 1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square.
- 1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.
- 1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.
- 1860 CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.
- 1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.
- 1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.
- 1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.
- 1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Assistant Physician to King's College Hospital; Examiner in Anatomy at the University of London; 3, Warwick street, Cockspur street.
- 1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.
- 1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.
- 1852 CUTLER, THOMAS, M.D., Spa, Belgium.
- 1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 1.
- 1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

Elected

- 1877 DARBISHIRE, SAMUEL DUKINFIELD, M.B., St. Bartholomew's Hospital.
- 1848 DAUBENY, HENRY, M.D., San Remo, Italy.
- 1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.
- 1853 DAVIES, ROBERT COKEB NASH, Rye, Sussex.
- 1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.
- 1876 DAVIES-COLLEY, JOHN NEVILLE C., M.C., Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 1.
- 1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; Examiner in Obstetric Medicine at the University of London; 24, Harley street, Cavendish square. C. 1869-70.
- 1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.
- 1878 DENT, CLINTON THOMAS, 29, Chesham Street, Belgrave square.
- 1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.
- 1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children; 9, Chesterfield street, Mayfair. C. 1874-5. *Trans.* 13. *Sci. Com.*
- 1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.
- 1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.

Elected

- 1845 DODD, JOHN.
- 1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to Out-Patients, Samaritan Hospital; Pathological Assistant to the Museum of the Royal College of Surgeons of England; 51, Seymour street, Portman square.
- 1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.
- 1867 DRAGE, CHARLES, M.D., Hatfield, Herts.
- 1853 DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Kensington Mall.] *Trans.* 2.
- 1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 17, Woburn place, Russell square.
- 1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. *Trans.* 1.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., 117, Cromwell road, South Kensington.
- 1845 DUFF, GEORGE, M.D., High street, Elgin.
- 1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.
- 1874 DUKA, THEODORE, M.D., [Surgeon-Major, H.M.'s Bengal Army]; 38, Montagu square.
- 1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.
- 1871 *DUKES, CLEMENT, M.D. and B.S., Horton crescent, Rugby, Warwickshire.
- 1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.
- 1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D. Ed., F.R.S. Ed., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square.

Elected

- 1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales ; 62, Pall Mall.
- 1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital ; 82, Brook street, Grosvenor square. C. 1876-7. *Trans.* 5. *Sci. Com.*
- 1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital ; 38, Brook street, Grosvenor square.
- 1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital ; Ipswich, Suffolk.
- 1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.
- 1836 EARLE, JAMES WILLIAM, late of Norwich.
- 1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary ; 69, Connaught street, Hyde park square.
- 1824 EDWARDS, GEORGE.
- 1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.
- 1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic ; 75, Harley street, Cavendish square.
- 1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle ; Coroner for Carlisle ; 35, Lowther street, Carlisle.
- 1848 ELLIS, GEORGE VINEY, late Professor of Anatomy in University College, London. C. 1868-4. *Trans.* 2.
- 1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.
- 1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor ; 14, High street, Windsor.
- 1842 †ERICHSEN, JOHN ERIC, F.R.S., Surgeon Extraordinary to H.M. the Queen ; Emeritus Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital ; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.

Elected

- 1874 EVANS, GEORGE HENRY, M.D.
- 1877 EWART, WILLIAM, M.B., 33, Curzon street, May Fair.
- 1875 *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.
- 1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; 11, St. Thomas's street, Southwark. *Trans.* 6.
- 1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.
- 1858 FALCONER, RANDLE WILBRAHAM, M.D., Consulting Physician to the Royal United Hospital, Bath; 22, Bennett street, Bath.
- 1862 FARQUHARSON, ROBERT, M.D., Assistant Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; Physician to the Belgrave Hospital for Children; 23, Brook street, Grosvenor square.
- 1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Sci. Com.*
- 1872 FAYRE, SIR JOSEPH, K.S.I., M.D., F.R.S. Edin., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 16, Granville place, Portman square.
- 1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; Chilton Hall, Ferry hill, and 16, Old Elvet, Durham.
- 1863 FENWICK, SAMUEL, M.D., Physician, with charge of Outpatients, to, and Lecturer on Medicine at, the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.
- 1852 *FIELD, ALFRED GEORGE.
- 1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

Elected

- 1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., Junior Physician to the West London Hospital; 92, Wimpole street, Cavendish square.
- 1878 FISHER, FREDERIC RICHARD, Assistant Surgeon to the Victoria Hospital for Sick Children; 79, Grosvenor street.
- 1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major. [6, Whitehall yard.]
- 1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.
- 1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 7, Waterloo street, Birmingham. *Trans.* 1.
- 1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.
- 1877 FONMARTIN, HENRY DE, M.D., House Physician, Seamen's Hospital, Greenwich; 474, King's Road, Chelsea, S.W.
- 1848 †FORBES, JOHN GREGORY, 82, Oxford terrace, Hyde park. C. 1868-9. *Trans.* 3.
- 1852 †FORSTER, JOHN COOPER, *Vice-President*, Surgeon to Guy's Hospital; Examiner in Surgery at the University of London; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-78. *Pro.* 1.
- 1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; Sydney, New South Wales.
- 1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.
- 1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.
- 1877 FOX, TILBURY, M.D., Physician to the Skin Department of University College Hospital; 14, Harley street.

Elected

- 1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the University of London; 67, Grosvenor street. C. 1875-6. *Trans.* 3.
- 1871 FRANK, PHILIP, M.D., Cannes, France.
- 1843 FRASER, PATRICK, M.D. C. 1866.
- 1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.
- 1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, Maida hill. C. 1852-3.
- 1876 FURNER, WILLOUGHBY, 111, King's road, Brighton.
- 1864 *GAIRDNER, WILLIAM TENNANT, M.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.
- 1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 2.
- 1865 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. *Trans.* 2.
- 1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.
- 1867 GARLIKE, THOMAS W., Malvern Villa, Churchfield road, Ealing.
- 1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.
- 1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 1.

Elected

- 1819 GAULTER, HENRY.
- 1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874-5.
- 1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Trans.* 1.
- 1821 *GEORGE, RICHARD FRANCIS, 1, South Parade, Weston-super-Mare.
- 1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy at University College; 22, Henrietta street, Cavendish square.
- 1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; 8, Upper Brook street, Grosvenor square.
- 1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.
- 1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Trans.* 2.
- 1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; 93, Gower street, Bedford square.
- 1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine at University College, and Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 4.
- 1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury Square.
- 1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Heathfield, Ringwood, Hants. C. 1863.

Elected

- 1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square.
- 1875 GREENE, WILLIAM T., M.A., M.D., 218, Old Kent road.
- 1875 GREENFIELD, W. S., M.D., Assistant-Physician to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 15, Palace road, Albert Embankment.
- 1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2. *Trans.* 1.
- 1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. C. 1876-7. *Trans.* 3.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.
- 1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.
- 1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris.
- 1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Trans.* 4.
- 1837 GULLY, JAMES MANBY, M.D.
- 1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.
- 1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

Elected

- 1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.
- 1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital, and to the Royal Westminster Ophthalmic Hospital; Standen House, Chute, Wilts. C. 1851. V.P. 1869.
- 1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; 16, Sussex place, Onslow gardens, and Duff House, Banff, N.B.
- 1836 HARDING, JOHN FOSSE, Ulverstone House, Uckfield, Sussex. C. 1858-9.
- 1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.
- 1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.
- 1864 HARLEY, JOHN, M.D., F.L.S., Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. *Trans.* 8.
- 1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.
- 1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.
- 1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.
- 1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.
- 1854 HAVILAND, ALFRED, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.
- 1870 HAWARD, J. WARRINGTON, Assistant-Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 5, Montagu street, Portman square. *Trans.* 1.

Elected

- 1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.
- 1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.
- 1848 HAWKESLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 17, Cheyne walk, Chelsea.
- 1875 HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-Accoucheur and Assistant-Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878.
- 1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.
- 1848 *HEALE, JAMES NEWTON, M.D., Medecroft, Winchester, Hants.
- 1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square.
- 1850 HEATON, GEORGE, M.D., Boston, U.S.
- 1874 *HEATON, JOHN DEAKIN, M.D., Senior Physician to the Leeds General Infirmary, and Lecturer on Medicine at the Leeds School of Medicine; Claremont, Leeds.
- 1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.
- 1877 HERMAN, GEORGE ERNEST, M.B.C.P., Assistant Obstetric Physician to the London Hospital; 20, Finsbury square.
- 1877 HERON, GEORGE ALLAN, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.

Elected

- 1843 HEWETT,[†] PRESCOTT GARDNER, F.R.S., Serjeant-Surgeon-Extraordinary to H.M. the Queen ; Surgeon in Ordinary to H.R.H. the Prince of Wales ; Consulting Surgeon to St. George's Hospital ; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris ; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans. 7. Sci. Com.*
- 1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital ; 36, Berkeley square. C. 1876.
- 1872 HEYN, JULIUS CHARLES WILLIAM, M.D., 42, Westbourne terrace, Hyde park.
- 1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to Guy's Hospital ; 38, Brook street, Grosvenor square.
- 1862 HILL, M. BERKELEY, M.B. Lond., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital ; Surgeon to the Lock Hospital ; 55, Wimpole street, Cavendish square. C. 1878.
- 1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.
- 1859 HIRD, FRANCIS, Senior Surgeon to the Charing Cross Hospital ; 13, Old Burlington street.
- 1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight ; Clifton House, Cowes, Isle of Wight.
- 1872 HOGG, FRANCIS ROBERTS, M.D., India.
- 1843 †HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital ; Consulting Surgeon to the Metropolitan Dispensary ; Surgeon to the Foundling Hospital ; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874.
- 1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital ; 10, Old Steyne, Brighton.
- 1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.

Elected

- 1856 HOLMES, TIMOTHY, M.A. Camb., *Secretary*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878. *Trans.* 6. *Sci. Com.*
- 1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.
- 1846 †HOLTHOUSE, CARSTEN, 15, George street, Hanover square, and Balham hill house. C. 1863.
- 1878 HOOD, DONALD WILLIAM CHARLES, M.B., 43, Green street, Park lane.
- 1878 HOUGHTON, WALTER B., M.D., Assistant Physician to Charing Cross Hospital; 26, Cavendish square.
- 1865 HOWARD, BENJAMIN, M.D., late Lecturer on Operative Surgery, and Surgeon to the Long Island College Hospital, New York. [Scientific Club, 7, Savile row].
- 1865 HOWARD, EDWARD, M.D.
- 1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's street, Southwark. *Trans.* 2.
- 1877 *HUDSON, ROBERT SAMUEL, M.D., 58, West-end, Redruth, Cornwall.
- 1857 HULKE, JOHN WHITAKER, F.R.S., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. *Trans.* 7. *Sci. Com.*
- 1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.
- 1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.
- 1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 5.

Elected

- 1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.
- 1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.
- 1856 HUTCHINSON, JONATHAN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. *Trans.* 7. *Pro.* 2.
- 1820 HUTCHINSON, WILLIAM, M.D.
- 1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.
- 1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.
- 1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.
- 1856 INGLIS, CORNELIUS, M.D., Athenæum Club, Pall Mall.
- 1876 IRVINE, JAMES PEARSON, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital; 3, Mansfield street, Cavendish square.
- 1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.
- 1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.
- 1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.
- 1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.
- 1825 JAMES, JOHN B., M.D.
- 1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

Elected

- 1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Trans.* 3.
- 1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Gènevois."
- 1847 JOHNSON, GEORGE, M.D., F.R.S., *Librarian*, Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. L. 1878. *Trans.* 10.
- 1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.
- 1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.
- 1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square. C. 1878. *Trans.* 5.
- 1876 JONES, LESLIE, M.D., 3, Brighton Parade, Blackpool, Lancashire.
- 1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]
- 1837 †JONES, THOMAS WILLIAM, M.D., Bylocks, Enfield Highway. C. 1858.
- 1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.
- 1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

Elected

- 1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.
- 1872 KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex.
- 1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.
- 1847 KEYSER, ALFRED, King's Hill, Berkhamstead.
- 1877 *KHORY, RUSTOMJEE NASERWANJEE, L. Med. Bombay, 47, Minford Gardens, West Kensington Park.
- 1857 KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.
- 1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*
- 1876 *KOCH, EDWIN LAWSON, M.D., Principal, Medical School of Ceylon; Colombo, Ceylon. [Agents: Messrs. Henry S. King & Co., 65, Cornhill.]
- 1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.
- 1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.
- 1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square.
- 1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

Elected

- 1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1816 LAWRENCE, G. E.
- 1843 *LEACH, JESSE, Moss Hall, Heywood, Lancashire.
- 1868 LEABED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.
- 1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V. P. 1868-9. *Trans.* 13. *Pro.* 2. *Sci. Com.*
- 1877 LEESON, ARTHUR EDMUND, M.A., M.D., 45, Devonshire street, Portland Place.
- 1869 LEGG, JOHN WICKHAM, M.D., Lecturer on Pathological Anatomy, St. Bartholomew's Hospital; 47, Green street, Park lane. *Trans.* 2.
- 1836 LEIGHTON, FREDERICK, M.D.
- 1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; 16, Albemarle street, Piccadilly.
- 1806 LIND, JOHN, M.D.
- 1878 LISTER, JOSEPH, F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.
- 1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.
- 1871 LITTLE, LOUIS STROMEYER, Shanghai, China.
- 1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.
- 1819 LLOYD, ROBERT, M.D.
- 1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.
- 1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

Elected

- 1836 LÖWENFELD, JOSEPH S., M.D., Berbice.
- 1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.
- 1877 LOWNE, BENJAMIN THOMPSON, 49, Colville gardens, Bayswater, W.
- 1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.
- 1857 LYON, FELIX WILLIAM, M.D., 49, Albany street, Leith, near Edinburgh.
- 1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.
- 1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.
- 1867 MAC CORMAC, WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.
- 1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.
- 1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.
- 1866 MACGOWAN, ALEXANDER THORBURN, Kingswood park, near Bristol.
- 1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.
- 1822 MACINTOSH, RICHARD, M.D.
- 1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.
- 1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.
- 1876 MACKEY, EDWARD, M.D., 22, Norfolk road, Brighton.
- 1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

Elected

- 1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.
- 1849 MACLURE, DUNCAN MACLACHLAN, M.B., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.
- 1876 MACNAMARA, CHARLES, Surgeon to the Westminster Hospital; Surgeon Major Bengal Medical Service; late Examiner in Surgery at the Calcutta University; 13, Grosvenor street.
- 1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.
- 1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.
- 1855 MARCET, WILLIAM, M.D., F.R.S.; Villa Bianca, Cannes. [30, Half Moon street]. C. 1871. *Trans.* 3. *Sci. Com.*
- 1867 MARSH, F. HOWARD, Assistant-Surgeon to St. Bartholomew's Hospital; Assistant Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. *Trans.* 2.
- 1838 MARSH, THOMAS PARR, M.D.
- 1851 MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-76. *Trans.* 2.
- 1864 MASON, FRANCIS, Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans.* 1.
- 1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.
- 1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square.
- 1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpelier row, Blackheath, Kent.
- 1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.

Elected

- 1874 MERRIMAN, JOHN J., 45, Kensington square.
- 1815 MEYER, AUGUSTUS, M.D., St. Petersburg.
- 1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.
- 1854 MIDDLESCHIP, EDWARD ARCHIBALD.
- 1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.
- 1844 †MONTEFIORE, NATHANIEL, 18, Portman square.
- 1836 MOORE, GEORGE, M.D., Hastings.
- 1873 MOORE, NORMAN, M.D., Warden of the College and Lecturer on Comparative Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.
- 1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.
- 1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.
- 1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.
- 1878 MORGAN, JOHN HAMMOND, M.A., 12, Chapel street, Park lane.
- 1874 MORRIS, HENRY, M.A. Lond., Senior Assistant-Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Trans.* 2.
- 1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.
- 1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans.* 1.

Elected

- 1856 MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital, Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 79, Wimpole street, Cavendish square. C. 1870-71. L. 1877. *Trans.* 3.
- 1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 29, Queen Anne street, Cavendish square.
- 1873 MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntress Row, Scarborough.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878.
- 1876 NAPIER, WILLIAM DONALD, 22, George street, Hanover square, W.
- 1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.
- 1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.
- 1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; 4, Wimpole street, Cavendish square.
- 1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.
- 1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.
- 1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.
- 1847 *NOURSE, WILLIAM EDWARD CHARLES, late Surgeon to the Brighton Children's Hospital; 11, Marlborough place, Brighton.
- 1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

Elected

- 1847 O'CONNOR, THOMAS, March, Cambridgeshire.
- 1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting-Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Trans.* 4.
- 1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, and The Elms, Derby.
- 1860 OGLE, WILLIAM, M.D., Medical Officer of Health for East Hertfordshire; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Trans.* 4.
- 1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].
- 1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.
- 1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 4.
- 1877 ORMEROD, JOSEPH ARDERNE, M.B., Upper Wimpole street.
- 1875 OSBOEN, SAMUEL, ; 17, Gresham park, Brixton, and 10, Maddox street, Regent street.
- 1874 PAGE, HERBERT WILLIAM, M.A., M.C. Cantab., Assistant Surgeon to, and Lecturer on Operative and Practical Surgery at, St. Mary's Hospital; 28, New Cavendish street. *Trans.* 1.
- 1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.
- 1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. P. 1875-76. *Trans.* 11. *Sci. Com.*
- 1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.
- 1847 PARKER, NICHOLAS, M.D., Paris.

Elected

- 1873 PARKER, ROBERT WILLIAM, Assistant-Surgeon East London Children's Hospital; 8, Old Cavendish-street. *Trans.* 1.
- 1841 PARKIN, JOHN, M.D. Rome; 5, Codrington place, Brighton.
- 1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 35, Grosvenor street.
- 1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 78, Wimpole street.
- 1845 †PEACOCK, THOMAS BEVILL, M.D., Consulting Physician to St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.
- 1856 PEIRCE, RICHARD KING, 94, Addison road, Kensington.
- 1830 PELECHIN, CHARLES P., M.D., St. Petersburg.
- 1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 18, Temple row, Birmingham. *Trans.* 1.
- 1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.
- 1870 PERRIN, JOHN BESWICK, Medical Tutor and Demonstrator of Practical and Surgical Anatomy, Owen's College; 51, Nelson street, Manchester.
- 1878 *PHILIPSON, GEORGE HARE, M.D., M.A. Cantab., Professor of Medicine at Durham University; 17, Eldon square, Newcastle-upon-Tyne.
- 1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater. C. 1877.
- 1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]
- 1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 13, South Eaton place, Eaton square. *Sci. Com.*

Elected

- 1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1.
- 1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.
- 1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.
- 1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.
- 1871 POORE, GEORGE VIVIAN, M.D., Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Trans.* 1.
- 1843 POPE, CHARLES, M.D., The Rectory, East Harptree, Bristol.
- 1846 POTTER, JEPHSON, M.D., F.L.S.
- 1842 POWELL, JAMES, M.D.
- 1867 POWELL, RICHARD DOUGLAS, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; Assistant-Physician to the Middlesex Hospital; 15, Henrietta street, Cavendish square. *Trans.* 1.
- 1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*
- 1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Sci. Com.*
- 1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 1, Pembridge place, Bayswater.

Elected

- 1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 9, Upper Wimpole street, Cavendish square. *Trans.* 2.
- 1878 PYE, WALTER, 4, Sackville street, Piccadilly.
- 1877 PYE-SMITH, PHILIP HENRY, M.D., Assistant-Physician to, and Lecturer on Physiology at, Guy's Hospital; 56, Harley street, Cavendish square.
- 1850 QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*
- 1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.
- 1852 RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.
- 1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.
- 1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.
- 1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital; the Pavement, Nottingham.
- 1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Peter-sham terrace], Queen's gate, South Kensington.
- 1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.
- 1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

Elected

- 1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.
- 1869 REEVES, WILLIAM, 5, the Crescent, Carlisle.
- 1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870.
- 1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.
- 1852 RICHARDSON, CHRISTOPHER THOMAS, M.B.
- 1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.
- 1863 RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Examiner in Materia Medica in the University of London; 15, Cavendish place, Cavendish square. *Trans.* 4.
- 1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.
- 1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.
- 1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Assistant Physician to University College Hospital; 53, Harley street, Cavendish square, W.
- 1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.
- 1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.
- 1843 RODEN, WILLIAM, M.D., Morningside, Kidderminster, Worcestershire.

Elected

- 1850 ROPE, GEORGE, M.D., Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge Road; 6, West street, Finsbury circus.
- 1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.
- 1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.
- 1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.
- 1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 91, New Hall street, Birmingham.
- 1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh.
- 1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.
- 1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3.
- 1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Professor of Physiology at the University of London; Jodrell Professor of Human Physiology and Histology at University College, London; 26, Gordon square, W.C. C. 1869-70. *Trans.* 2. *Sci. Com.* 2.
- 1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.
- 1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Andoversford, Cheltenham.
- 1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; 30, Devonshire street, Portland place. *Trans.* 2.
- 1845 †SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

Elected

- 1834 SAUVAN, LUDWIG V., M.D., Warsaw.
- 1859 SAVORY, WILLIAM SCOVELL, F.R.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. *Trans.* 4. *Sci. Com.* 3.
- 1873 SCOTT, J. M. JOHNSTON, M.D., 14, College square, east, Belfast.
- 1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.
- 1877 SEATON, EDWARD CATOR, M.D., Medical Officer of the Local Government Board; 14, Gordon street, Gordon square.
- 1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. *Trans.* 2.
- 1877 SEMON, FELIX, M.D., 6, Chandos street, Cavendish square.
- 1875 SEMPLÉ, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square.
- 1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.
- 1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.
- 1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London; 50, Torrington square. C. 1848-9. V.P. 1862.
- 1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.
- 1859 SIBLEY, SEPTIMUS WILLIAM, 4, Savile row. *Trans.* 4. *Sci. Com.*
- 1848 SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Trans.* 2. *Sci. Com.*

Elected

- 1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.
- 1842 †SIMON, JOHN, C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.
- 1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.
- 1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.
- 1872 SMITH, GILBERT, M.A., M.B., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; Visiting Physician to the Margaret Street Infirmary for Consumption; 68, Harley street, Cavendish square.
- 1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.
- 1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.
- 1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Trans.* 3. *Sci. Com.*
- 1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.
- 1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.
- 1874 *SMITH, WILLIAM ROBERT, M.B., Hon. Med. Officer, Sheffield Hospital for Sick Children, Broomhill, Sheffield.
- 1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

Elected

- 1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square. *Trans* 1.
- 1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.
- 1874 SPARKS, EDWARD ISAAC, M.B. [Abroad.] *Trans*. 1.
- 1851 SPITTA, ROBERT JOHN, M.D. Lond., Clapham Common, Surrey. C. 1878. *Trans*. 1.
- 1875 SPITTA, EDMUND J., late Demonstrator of Anatomy at St George's Hospital; Ivy House, Clapham Common, Surrey.
- 1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.
- 1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.
- 1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.
- 1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1865 STOKES, WILLIAM, M.D., Professor of Surgery, Royal College of Surgeons, Ireland, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin. *Trans*. 1.
- 1843 STORKS, ROBERT REEVE, Paris.
- 1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5.
- 1876 STRETTON, WILLIAM HARRIS, M.D., Physician to the Farringdon Dispensary; 8, Suffolk place, Pall Mall East.
- 1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.
- 1863 STURGES, OCTAVIUS, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children, Wimpole street, Cavendish square. C.

Elected

- 1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.
- 1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.
- 1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Medicine at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.
- 1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.
- 1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.
- 1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.
- 1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.
- 1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.
- 1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.
- 1875 TAY, WARREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.
- 1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark.
- 1852 TAYLOR, ROBERT, 7, Lower Seymour street, Portman square.
- 1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.
- 1859 TEGART, EDWARD, 49, Jermyn street, St. James's.
- 1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 4.

Elected

- 1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878. *Trans.* 1. *Sci. Com.*
- 1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.
- 1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 5.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 9, Cranley place, South Kensington. *Trans.* 2. *Sci. Com.*
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 83, Park street, Grosvenor square. *Trans.* 1.
- 1873 TIBBITS, HERBERT, F.R.C.P. Ed., Medical Superintendent of the National Hospital for the Paralysed and Epileptic; 30, New Cavendish street.
- 1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square.
- 1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square.
- 1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.
- 1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.
- 1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.
- 1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

Elected

- 1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital ; 15, Finsbury square.
- 1873 TURNER, GEORGE BROWN, M.D., 3, Warrior square, St. Leonard's-on-Sea.
- 1876 VENN, ALBERT JOHN, M.D., C.M., Obstetric Physician to the Metropolitan Free Hospital ; Assistant Physician to the Victoria Hospital for Children ; 40, Brook street, Grosvenor square.
- 1870 VENNING, EDGCOMBE, late Surgeon, 1st Life Guards ; 87, Sloane street.
- 1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital ; 43, Weymouth street, Portland place.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square ; 141, Regent street.
- 1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.
- 1854 WADDINGTON, EDWARD, Auckland, New Zealand.
- 1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital ; 14, Park lane.
- 1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary ; 3, Old Burlington street.
- 1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary ; 25, Lowther street, Carlisle.
- 1867 *WALLIS, GEORGE, Corpus Buildings, Cambridge.
- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital ; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road ; 27, Weymouth street, Portland place.
- 1852 WALSH, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London ; Consulting Physician to the Hospital for Consumption ; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

Elected

- 1851 WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.
- 1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.
- 1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.
- 1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.
- 1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.
- 1818 WARE, JOHN, Clifton Down, near Bristol.
- 1866 WARING, EDWARD JOHN, M.D., 49, Clifton gardens, Maida vale.
- 1877 WARNER, FRANCIS, M.D., 15, Finsbury square.
- 1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.
- 1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.
- 1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square. *Trans.* 1.
- 1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.
- 1840 WEBB, WILLIAM WOODHAM, M.D.
- 1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

Elected

- 1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Trans.* 6.
- 1844 †WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-8.
- 1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualaguay-chu, Entre Rios, Argentine Confederation.
- 1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row. C. 1877.
- 1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; late Professor of Surgery and Pathology at the Royal College of Surgeons; 3, Upper Grosvenor street. C. 1870. *Trans.* 10. *Pro.* 1.
- 1842 †WEST, CHARLES, M.D., *President*, Corresponding Member of the Academy of Medicine of Paris; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Trans.* 2. *Sci. Com.*
- 1877 WEST, SAMUEL, M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 8, Guilford street, Russell square.
- 1828 WHATLEY, JOHN, M.D.
- 1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Pathology at, St. George's Hospital; 37, Green street, Grosvenor square.
- 1849 WHITE, JOHN.
- 1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.
- 1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.
- 1844 †WILDBORE, FREDERIC, 245, Hackney road.
- 1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.
- 1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

Elected

- 1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.
- 1865 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.
- 1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.
- 1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Trans. 1. Sci. Com.*
- 1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.
- 1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Trans. 3.*
- 1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square.
- 1859 WILLIAMS, JOSEPH, M.D. Holmhurst, Cambridge park, Twickenham.
- 1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; Bethlehem Royal Hospital, Lambeth road.
- 1839 †WILSON, ERASMUS, F.R.S., late Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. C. 1877. *Trans. 2.*
- 1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.
- 1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

Elected

- 1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill,
Upper Norwood, Surrey.
- 1841 WOOD, GEORGE LEIGHTON, 28, Green park, Bath.
- 1851 WOOD, JOHN, F.R.S., *Vice-President*, Senior Surgeon to
King's College Hospital, and Professor of Clinical Sur-
gery in King's College, London; 68, Wimpole street.
C. 1867-8. V.P. 1877-8. *Trans.* 3.
- 1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.
- 1848 WOOD, WILLIAM, M.D., *Vice-President*, Physician to St.
Luke's Hospital for Lunatics; 99, Harley street, Caven-
dish square. C. 1867-8. V.P. 1877-8.
- 1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft,
Suffolk. *Trans.* 3.
- 1865 WOTTON, HENRY, M.D., 62, Bedford gardens, Kensington.
- 1878 YEO, GERALD F., M.D., M.Ch., Demonstrator of Surgery at
King's College, London; 15, Albemarle street, Pic-
cadilly.

[It is particularly requested that any change of Title, Appointment, or
Residence, may be communicated to the Secretaries before the 1st of
October in each year, in order that the List may be made as correct as
possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

- 1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.
- 1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.
- 1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D., Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.
- 1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.
- 1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.
- 1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; President of the Royal Society; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.
- 1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

Elected

- 1847 OWEN, RICHARD, C.B., D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum ; Foreign Associate of the Academy of Sciences of the Institute of France ; Sheen Lodge, Mortlake.
- 1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge ; Secretary to the Royal Society, &c. ; Lensfield Cottage, Cambridge.
- 1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution ; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c. ; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

- 1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna ; Vienna.
- 1864 DONDEES, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
- 1875 DRAPER, JOHN WILLIAM, M.A., LL.D., Emeritus Professor of Chemistry and Physiology in the University of New York ; 13, University Buildings, Washington square, New York.
- 1876 EDWARDS, H. MILNE, M.D., Member of the Institute of France, and of the Academy of Medicine ; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris ; 57, Rue Cuvier, Paris.
- 1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden ; President of the College of Health, and Director-General of Hospitals ; Stockholm.
- 1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., LL.D., Professor of Surgery in the Jefferson Medical College of Philadelphia.
- 1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.
- 1873 HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics ; Berlin.
- 1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.
- 1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.
- 1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

Elected

- 1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.
- 1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France ; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées ;" Commander of the Legion of Honour, &c. ; Rue de Lille, 91, Paris.
- 1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute ; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena ; St. Petersburg.
- 1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin ; Corresponding Member of the Academy of Sciences of the Institute of France ; Berlin.

CONTENTS.

	PAGE
List of Officers and Council	v
Presidents of the Society	vi
Referees of Papers	vii
Trustees of the Society	viii
Library Committee	viii
List of Fellows	ix
List of Honorary Fellows	lvii
List of Plates	lxv
Woodcuts	lxvi
Advertisement	lxvii
Regulations relative to Proceedings	lxviii

<p>I. On a Case of Noma, in which Moving Bodies were observed in the Blood during Life. By ARTHUR ERNEST SANSOM, M.D.Lond., F.R.C.P., Senior Physician to the North-Eastern Hospital for Children, and Assistant Physician to the London Hospital</p>	1
<p>II. On a large Aneurism of the Aorta, Innominate, Subclavian, and Carotid Arteries, treated successfully by Double Distal Ligature. By RICHARD BARWELL, F.R.C.S., Surgeon to the Charing Cross Hospital</p>	13
<p>III. On the Removal by Operation of a Hairy Mole occupying half the Forehead. By W. MORRANT BAKER, F.R.C.S., Lecturer on Physiology and Assistant Surgeon to St. Bartholomew's Hospital, and Senior Surgeon to the Evelina Hospital for Sick Children</p>	33
<p>IV. Cases of Branchial Fistulæ in the External Ears. By Sir JAMES PAGET, Bart., D.C.L., LL.D., F.R.S.</p>	41

	PAGE
V. The Microscopic Anatomy of the Smooth Tongue, "Chronic Superficial Glossitis." By HENRY TRENTHAM BUTLIN, F.R.C.S., Surgical Registrar to St. Bartholomew's Hospital, Assistant Surgeon to the West London Hospital	51
VI. On Myxœdema, a term proposed to be applied to an essential condition in the "Cretinoid" Affection occasionally observed in Middle-aged Women. By WILLIAM M. ORD, M.D. Lond., F.R.C.P., Physician to St. Thomas's Hospital	57
VII. On the Pathology of Tetanus and Hydrophobia. By JOSEPH COATS, M.D., Lecturer on Pathology and Pathologist to the Western Infirmary, Glasgow. (Communicated by Dr. W. T. GAIRDNER.)	79
VIII. On the Proportion of Red Corpuscles in the Blood in some Skin Diseases. By GEORGE THIN, M.D.	95
IX. A Second Communication on Simple Atrophic Sclerema. By JOHN HARLEY, M.D. Lond., Senior Assistant Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital	101
Appendix to ditto, Post-mortem, &c.	313
X. Analysis of Seventy-five Cases of "Writer's Cramp" and Impaired Writing Power. By GEORGE VIVIAN POORE, M.D., F.R.C.P., Assistant Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women	111
XI. On a Case of Amnesia, with Post-mortem Examination. By WILLIAM HENRY BROADBENT, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital	147
XII. An Account of 500 Cases of Operation for Stone in the Bladder of the Male Adult, comprising his entire experience of such cases to January, 1877, with Remarks on the most important Incidents in connection with them. By Sir HENRY THOMPSON, F.R.C.S., Surgeon Extraordinary to H.M. the King of the Belgians, Consulting Surgeon to University College Hospital, and Emeritus Professor of Clinical Surgery at University College, London	159

	PAGE
XIII. On the Condition of the Skin in <i>Tinea tonsurans</i> . By GEORGE THIN, M.D.	179
XIV. One Hundred Cases of Paracentesis of the Tympanic Membrane, with the Results obtained therefrom, and Remarks on the Methods of Operation. By W. LAIDLAW PURVES, M.D., Aural Surgeon to Guy's Hospital	189
XV. On some Points in the Minute Anatomy of the Kidney and their relation to the Pathological Phenomenon of Tubular Casts. By REGINALD SOUTHEY, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital	201
XVI. On Paralysis of the Internal Muscles of the Eye (Ophthalmoplegia Intima), a Group of Symptoms which probably indicates Disease of the Lenticular Ganglion. By JONATHAN HUTCHINSON, F.R.C.S., Senior Surgeon to the London Hospital, Surgeon to the Moorfields Ophthalmic Hospital, and to the Hospital for Skin Diseases	215
XVII. Treatment of Hæmorrhage from Punctured Wounds of the Throat and Neck, especially considered with regard to Ligature of the External Carotid Artery. By WM. HARRISON CRIPPS, F.R.C.S., Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital	229
XVIII. A Contribution to the Pathology of Hæmophilia. By PERCY KIDD, B.A. Oxon., Demonstrator of Practical Physiology at St. Bartholomew's Hospital. (Com- municated by Dr. SAMUEL GEE)	243
XIX. On the Pathological Traces of Pulmonary Hæmor- rhage. By REGINALD E. THOMPSON, M.D., F.R.C.P., Senior Assistant Physician to the Hospital for Con- sumption, Brompton, and Curator of the Patho- logical Museum	253
XX. Notes on the Spirillum Fever of Bombay, 1877. By H. VANDYKE CARTER, M.D. Lond., H.I.M. Indian Army. (Communicated by JOHN HARLEY, M.D.)	273
XXI. Case of Intussusception in which Abdominal Section was performed. By C. HANDFIELD JONES, M.B. Cantab., F.R.S., Physician to St. Mary's Hospital; and HERBERT W. PAGE, M.C. Cantab., F.R.C.S., Assistant Surgeon to St. Mary's Hospital	301

	PAGE
XXII. Appendix to Dr. JOHN HARLEY's Paper, page 101, on Simple Atrophic Sclerema : Sequel of Case of Wm. W—, Post-mortem, &c.	313
INDEX	317

LIST OF PLATES.

	PAGE
I. Case of Noma. Figs. 1—4. The Translucent Bodies in the Blood, and variations in size of the Red Blood-corpuscles. (A. E. SANSOM, M.D.)	12
II. Aneurism of Aorta. Figs. 1, 2, from Photographs taken immediately before, and seven weeks after, the Operation. (R. BARWELL)	32
II*. Ditto, Dissection of the Aneurism, Arch of Aorta, &c. (R. BARWELL)	32
III. Removal by Operation of a Hairy Mole. (W. MORRANT BAKER)	40
IV. Chronic Superficial Glossitis. Figs. 1—5. (H. T. BUTLIN)	56
V. Cases of Myxœdema. Figs. 1, 2. Sections of Eyelid. (WM. M. ORD, M.D.)	78
VI. Ditto. Fig. 1. Section of Kidney. Fig. 2. Section of Thyroid Body. (WM. M. ORD, M.D.)	78
VII. Ditto. Photographs. Fig. 1. Appearance of Patient at age of 21. Fig. 2. Appearance seven years later. (WM. M. ORD, M.D.)	78
VIII. Pathology of Tetanus and Hydrophobia. Figs. 1—5. Tetanus, from Sections of the Medulla Oblongata and the Convolutions. Fig. 7. Hydrophobia, from Section of Spinal Cord. (JOS. COATS, M.D.)	94
IX. Ditto. Figs. 7—10. Hydrophobia. Sections of the Medulla Oblongata, and from middle of Cervical Region, leucocytes. Figs. 11, 12. Rabies in Dog. Sections of Parotid Gland and Kidney. (JOS. COATS, M.D.)	94
X. Simple Atrophic Sclerema. (JOHN HARLEY, M.D.)	110
XI. Cases of Writer's Cramp. (G. V. POORE, M.D.)	146
XII. The Skin in Tinea tonsurans. Figs. 1—8. (GEORGE THIN, M.D.)	188
XIII. Anatomy of the Kidney. Figs. 1—4. (REGINALD SOUTHEY, M.D.)	214
XIV. Pathology of Hæmophilia. Figs. 1—6. (PERCY KIDD, B.A.)	252
XV. Pathological Traces of Pulmonary Hæmorrhage. Figs. 1—3. (REGINALD E. THOMPSON, M.D.)	272
XVI. Ditto. Ditto.	272
VOL. LXI.	e

LIST OF PLATES—*continued*.

	PAGE
XVII. The Spirillum of Recurrent Fever. Figs. *, and a to z. (VANDYKE CARTER, M.D.)	300
XVIII. Ditto, Figs. 1—12. (VANDYKE CARTER, M.D.)	300

WOODCUTS.

Case of Noma: Chart of Temperature, Pulse, and Respiration. (A. E. SANSOM, M.D.)	4
Aneurism of the Aorta, Innominate, Subclavian, and Carotid Arteries, treated by double distal ligature; tape and compass measurement of the tumour. (R. BARWELL)	16
Ditto, Chart of Temperature and Pulse. (R. BARWELL)	21
Branchial Fissure on the Helix of the Ear. (Sir JAMES PAGET)	49
Pathological Traces of Pulmonary Hæmorrhage, Fig. 1. (REGINALD E. THOMPSON, M.D.)	258
Ditto, Fig. 2. (REGINALD E. THOMPSON, M.D.)	259

TABLES.

Writer's Cramp: Analysis of Cases. (G. V. POORE, M.D.)—	
Table 1, Group I—IV	112
„ 2, „ IV, V	128
„ 3, „ V, VI	128

ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its Transactions.

REGULATIONS relative to the publication of the 'Proceedings
of the Society.'

That, as a general rule, the 'Proceedings' will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the 'Proceedings' will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That the 'Proceedings of the Society' may be obtained by non-members at the Society's House, 53, Berners Street, on prepayment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps;—this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the Proceedings *only*, or in the Proceedings and Transactions; and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ON A CASE OF NOMA,
IN WHICH
MOVING BODIES WERE OBSERVED IN THE
BLOOD DURING LIFE.

BY
ARTHUR ERNEST SANSOM, M.D. LOND., F.R.C.P.,
SENIOR PHYSICIAN TO THE NORTH-EASTERN HOSPITAL FOR CHILDREN AND
ASSISTANT PHYSICIAN TO THE LONDON HOSPITAL.

(Received October 2nd—Read October 9th, 1877.)

THE history of the following case presents several points of interest. These points are chiefly (1) the discovery in the blood and excretions, during life, of minute translucent bodies in active movement—Noma is therefore added to the list of diseases in which organisms, or quasi-organisms, may be present in the fluids; (2) the observations that these bodies had peculiar characteristics, and have been probably hitherto undescribed; and (3) the demonstration that the diseased structures, as well as the blood, possessed the faculty of virulent infectiveness, inoculation of the blood inducing the manifestation of motile particles similar to those which itself contained.

It will be convenient to divide this communication into three sections, comprising—I. A general history of the case. II. An account of the microscopical examination of the blood. III. A record of inoculation experiments upon animals.

At the outset I have to express my indebtedness to Mr. J.
VOL. LXI.

Needham, late House Surgeon to the North Eastern Hospital for Children, for the painstaking manner in which he observed and recorded the case.

I. *General history*.—Alice C—, aged four years and three months, was admitted an in-patient of the North Eastern Hospital for Children, under the care of Dr. Sansom and Mr. Waren Tay, on February 12th, 1876. She had enjoyed good health until a fortnight previously, when she complained of pain in the left cheek, which soon became swollen. A chemist was consulted, who pronounced the disease a "gumboil" of trivial consequence. The child became progressively worse, until she was brought to the hospital as an out-patient, and was at once admitted to the wards.

On admission there was found to be great prostration; the child very drowsy, though irritable, anæmic, but not emaciated, and presenting no signs of recent acute specific fever. Temperature 101.2° . Urine clear, sp. gr. 1015, reaction acid, no albumen. The left cheek was much swollen and indurated, except at one point mid-way between the angle of the mouth and the angle of the inferior maxilla, where a sensation of "bogginess" was communicated to the finger, and on pressure a quantity of very foetid, puriform fluid flowed from the mouth. The breath was extremely offensive, the lips parched and covered by a black sordes; there were no decayed teeth. Ordered milk, beef tea, and wine. A mixture of sulphocarbolate of sodium and sulphite of sodium (of each 10 grains) with Decoctum Cinchonæ. A gargle and wash of solution of carbolate of glycerine. Cataplasma Lini to wound.

February 13th.—On removing the poultice this morning a circle of integument of about the area of a half-crown piece was found to be of a brown colour and denuded of epidermis; this corresponded to the boggy region noticed yesterday. The necrosed tissue was removed, and the subjacent structures were found to be in a sloughy condition. On removing the slough a large cavity was disclosed, extending from the ramus of the inferior maxilla forwards nearly as far as the chin, and inwards beneath the tongue.

The tissues in front of the carotid were seen to move in unison with the pulsations of that vessel; the facial artery was quite obliterated; the surface of the inferior maxillary bone was bare and blackened; the parts exhaled a sickening odour. No pain was caused by the necessary manipulations. The cavity was plugged with strips of lint saturated in Liq. Ferri Perchlor. fort. (P. B.).

14th.—Cavity enlarging. Under Mr. Tay's superintendence, the actual cautery was freely used.

15th.—Cheek rapidly sloughing; slight œdema of left eyelids. Nourishment taken plentifully; no evidence of pain. Child is very quiet, but has frequent short, hacking cough. Physical examination shows dulness over the base of the right lung posteriorly, with increased vocal resonance and tactile vocal fremitus. Over scattered areas, bronchial breathing and fine crepitant râles. Some rhonchi were heard over both sides of the chest. At mid-day blood was seen to be flowing from the dressings and from the mouth; the hæmorrhage was very copious, and the child became extremely pale and almost pulseless, it was, however, promptly arrested.

16th.—There is some general œdema, the left hand being particularly swollen.

17th.—œdema increasing.

18th.—A canine and two bicuspid teeth fell out this morning. Sloughing extends to ear, orbit, and neck; the left temporo-maxillary articulation is laid bare, and there is lateral dislocation of the lower jaw; all muscular attachments have sloughed away from the left half of the bone, periosteum has disappeared, and the osseous tissue is dry and black. Face œdematous. Pulse very frequent, small, and feeble. No pain, but the child has great difficulty in swallowing, fluids escaping through the wound. Ordered Quinæ disulph. gr. v omni alterâ horâ. A draught containing Sp. Ammon. Arom. ℥xv, ter die.

19th.—The wound is now continuous with the cavity of the mouth, and ulceration is rapidly extending on all sides. Actual cautery applied to the margins; no pain evidenced.

20th.—Patient hardly conscious; respirations very rapid. Abundant mucous râles heard all over the chest. Frequent vomiting; food refused. Nutrient enemata were administered. The child gradually sank, and expired in the evening.

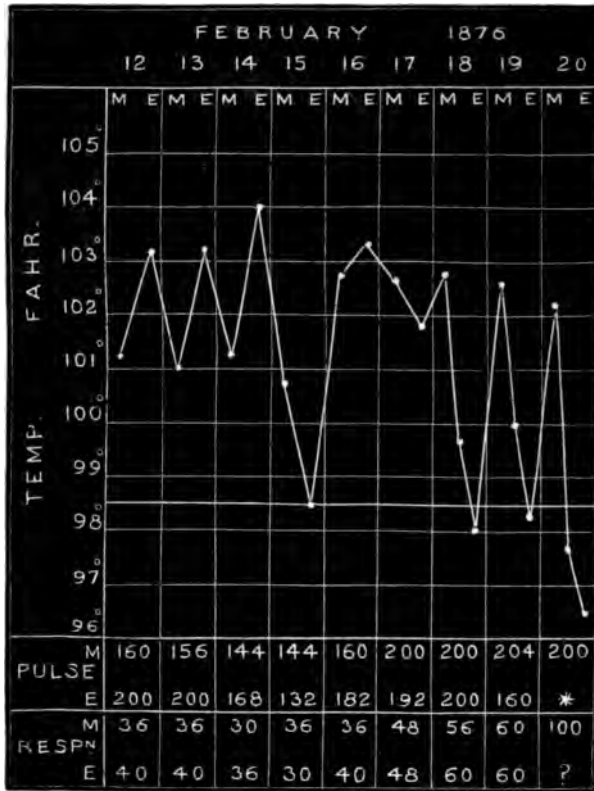


Chart of temperature, pulse, and respirations.

Autopsy 40 hours after death.—Body well nourished. Œdema general, and equally distributed.

Head.—A large excavation involves the left side of the face, it extends upwards to the orbit, forwards to within an inch of the nose, and is bounded externally and inferiorly by sloughy integument following pretty closely the borders

of the inferior maxilla. The external wound is $2\frac{1}{2}$ inches in diameter; its edges are raised and hard, and the neighbouring tissues are indurated. The superior and inferior maxillary bones, as well as the temporal, are laid bare. The left side of the tongue is in a sloughing condition, the temporal muscle has sloughed, and all the neighbouring structures present signs of necrosis; the vessels supplying the structures are for a short distance filled with firm clots.

The brain appears healthy, except that it is extremely anæmic.

Lungs.—There is general bronchitis. The basic portions are consolidated; on section a dirty brown fluid exudes; the surface of the section is more granular, and the pulmonary substance is softer than normal. Microscopical examination shows patches formed of alveoli, filled with round cells, contiguous to other alveoli which appear healthy—the usual signs of broncho-pneumonia.

Heart normal, containing blood-clot.

Liver anæmic, but no other morbid change, weight $22\frac{1}{2}$ oz.

Spleen, weight $1\frac{3}{4}$ oz., small and anæmic.

Kidneys anæmic, but in other regards healthy.

II. *Examination of the blood during life*.—Microscopical examination of the blood obtained from punctures of the fingers and the toes was first made on February 15th, the third day after the child's admission. It was at once seen that there was a considerable excess of the white elements. The coloured corpuscles very rapidly formed rouleaux with very large interspaces in which might be seen many colourless cells, which, before coming to rest, seemed to exhibit their amœboid movements with more than usual activity. The appearances conveyed the idea, not only that the white elements were in excess, but that the coloured elements were in deficient proportion. The protoplasm of the white elements seemed to be abnormally granular, and the fluid plasma was seen to contain fragmentary leucocytes and many minute masses of granular matter. The minute particles, when examined by a higher power (7 or 8 Hartnack), were

seen to consist of two varieties, *first*, granules so commonly seen in conditions of pyrexia; *secondly*, small highly refractile bodies endowed with powers of rapid locomotion. On account of such motion the exact shape of these bodies was difficult to determine, but, when comparatively still and a little out of focus, each one refracted light in such a manner that a small bright cross was visible in its substance. Thus they resembled crystals of oxalate of lime, with the exception that they were not perfect octahedra, but appeared as fragmentary crystalloids. (Plate I, fig. 1.)

The movement of these bodies resembled that of very minute bacteria. It was lineal and sometimes opposed a current in the fluid; thus, two or three which were observed to be attached to a red blood-corpuscle were seen to move the corpuscle in a direction opposite to that in which many other corpuscles immediately surrounding it were carried. It had no resemblance to Brownian or molecular movement; in fact, the two varieties of movement could be observed in the same field, portions of molecular matter being agitated in the "swarming" manner of inorganic particles, whilst the refractile bodies proceeded with greater velocity in a far less limited area, and described straight lines rather than circles. Occasionally one of the translucent bodies was seen to revolve many times upon its axis, and then suddenly to start away in a straight line with that apparently purposive progress which characterises some bacteria. The field having been watched for a few minutes, very fine fibrillæ were seen to start from the ordinary still particles of granular matter, and to extend in all directions; the meshes of the network thus formed enclosed the refractile bodies, at first limiting their movements, and afterwards bringing them almost, or completely, to rest (Plate I, fig. 2).

It was found that the addition of a two per cent. solution of carbolic acid to the blood during observation immediately caused the movements of the translucent particles to cease. At the same time it produced rapid changes in the normal elements, the coloured corpuscles assuming an appearance resembling that produced by a two per cent. solution of

tannic acid—the œcoïd and zöoid of Brücke. A solution of quinine also stopped the movements. On the other hand, heat induced an acceleration of the characteristic motion; a very dilute solution of potash also increased the movements, whilst the granular matter was dissolved and the corpuscles variously altered; a weak solution of sulphuric acid also stimulated the activity of the translucent bodies and caused some that were previously at rest to assume movement. These reagents did not destroy the translucent bodies nor change their outline.

The coloured blood-corpuscles showed strange variations in size (Plate I, fig. 3). Some measured $\frac{1}{3400}$ inch in diameter, whilst others were as small as $\frac{1}{8000}$, and between these extremes there were many gradations. The smaller corpuscles acted upon transmitted light exactly as the larger; their contours were circular and regular, and all the corpuscles were of the same tint. The protoplasm of the white elements appeared to be more diffuent than normal; there were small detached masses and granular débris.

Whilst the case was under observation, there was much variation in the proportion of the refractile bodies and in other conditions of the blood. At eight o'clock in the evening of the day (February 15th) on which the observations upon the blood were first made, the number of motile particles was notably diminished, at the same time it was seen that there was far less granular matter and fewer leucocytes. The red blood-corpuscles presented all variations in size. It is to be noted that at this time the temperature had fallen to 98.4° F., and considerable hæmorrhage had taken place. On the following day, temperature having risen nearly to 103° F., the characteristic translucent bodies were in great abundance. On occasions when this abundant presence of the particles was manifest, they were observed to become aggregated in groups resembling zoöglæa forms (Cohn) (Plate I, fig. 4). This grouping enabled us to estimate approximately the size of the particles. We found that about 20 occupied the space of a coloured blood-corpuscle of full size. At this time (February 16th) the colourless elements

were in excess, the smaller red corpuscles were not seen, and the blood did not coagulate so rapidly as on the previous day. The notable variations in size of the red blood-corpuscles were not again observed until February 19th, when they were as marked as on the first occasion.

On the 20th (day of decease), in addition to the translucent bodies, were seen many rod-shaped bodies having all the characters of ordinary bacteria. These were for the most part single, but a few were jointed. They insinuated themselves between the rows of blood-corpuscles, and when in a clear space proceeded rapidly with the motion characteristic of bacteria. They speedily left the field during observation, and were only followed by the stage of the microscope being moved.

The urine was examined immediately on being voided. On the first occasion in which the translucent bodies were discovered in the blood, particles having exactly similar characters were found also in the urine. For the most part these were isolated, but some adhered together in small groups resembling *sarcinæ*. Vibriones and ordinary bacteria became manifest after a short time, but these ordinary attendants upon decomposition were never observed, as the translucent bodies were, immediately after the urine was voided. On the 17th the fæces were examined and numbers of the characteristic bodies observed therein.

The discharges from the wound also evidenced large numbers of the translucent bodies, which were in active movement, together with multitudes of vibriones, bacteria, and the ordinary attendants upon putrefaction.

III. *Inoculation experiments upon animals.*—Specimens of (1) the blood collected at the autopsy from the right side of the heart ; (2) the fluid discharges from the wound were preserved in sealed capillary tubes.

On February 22nd a minute quantity of the blood from the right side of the heart was injected into the abdominal cavity of a healthy mouse, the animal being anæsthetized by chloroform. It recovered very rapidly and seemed perfectly

healthy. Next morning it was found very lethargic and it died at 10 a.m. On opening the abdominal cavity the small intestines were seen to be covered with a greyish, gelatinous material which, on microscopic examination, was found to contain pus corpuscles, bacteria and vibriones, and a large number of the translucent bodies, strictly resembling those described as existing in the case of the child. These bodies were also discovered in active motion in the blood of the animal, and with these were a few rod-shaped bacteria.

On February 22nd a few minims of the *blood* from the right side of the heart were injected into the abdominal cavity of a healthy guinea-pig, the animal being chloroformed. It promptly recovered, and was observed to be quite lively all the following day, taking plenty of food.

On the 26th a little bloody fluid was observed on the surface of the abdomen; it was then found that over a limited area the skin was extremely tender and soft, the hair coming off with the slightest touch. The animal died early in the morning of the 27th. On incising the abdominal wall after death it was seen that the softening extended through the whole thickness; all the tissues were infiltrated, but the condition merged circumferentially and gradually into healthy tissue; there was no line of demarcation. The tissues where infiltration commenced were much congested; those nearer the centre had their blood-vessels blocked with dark coagula. Microscopic examination of the fluid which saturated the diseased part disclosed broken-down muscular fibre, fat globules, a few crystals of phosphate and of oxalate of lime, a large number of large granular corpuscles, and innumerable bacteria, vibriones, and many chains of leptothrix.

Of the *heart*, the right side was greatly distended by a large dark clot; the right ventricle contained also a little fluid which showed, on microscopic examination, large numbers of blood-crystals and a few of the refractile bodies, some of which were at rest, while others were in active motion. No other forms of bacteria could be seen. The left ventricle was rather contracted, but contained a small black clot. The left auricle also contained a small black

clot, as well as a little fluid blood. This blood was seen to contain a number of the translucent bodies, as well as a few dumb-bell shaped bacteria (*bacterium termo*). Fluid blood drawn from the vena cava was seen to contain an enormous number of the translucent bodies ; the blood-corpuscles were unaltered.

The *lungs* were moderately congested ; scattered throughout them were minute patches of a dark reddish material, the nature of which was doubtful, but was probably an amorphous derivative of hæmoglobin. The other viscera presented no signs calling for note.

A minute quantity of the fluid obtained from the sloughing tissues about the *diseased portion of the mouth* of the child was injected into the abdominal cavity of a healthy mouse on February 22nd, 1876, the mouse being under chloroform. The mouse recovered and appeared quite lively, but was found dead the next morning. On *post-mortem examination* nothing abnormal could be detected.

A few minims also obtained from the fluid of the diseased part in the child were injected into the abdominal cavity of a large and healthy cat. The cat recovered well from anaesthesia and appeared quite well the next morning ; during the following day, however, the animal took very little food, and appeared very lethargic, though no signs of pain were evinced. The animal was found dead on the eighth morning after the injection ; it had shown signs of pain for one or two days previously. On *post-mortem examination* the abdomen was found to be filled with foetid, greenish-yellow pus, and there were the usual signs of intense peritonitis. In the other organs of the body no morbid signs were discovered, and examination of the blood evidenced nothing abnormal.

So, far, therefore, as these experiments permit a conclusion it would appear that whilst inoculation of the fluids derived from the diseased tissue (with the decomposing matters which they necessarily contained) induced peritonitis without discoverable alteration of the blood, inoculation

WERE OBSERVED IN THE BLOOD DURING LIFE. 11

of the diseased *blood* produced septicæmia with the manifestation of the characteristic motile particles observed in the original disease.

DESCRIPTION OF PLATE I.

Case of Noma. (A. E. Sansom, M.D.)

FIG. 1.—Blood examined immediately after puncture of the finger. The translucent bodies in some instances show the bright cross produced by refraction. The white elements are more than usually irregular in outline; the red elements, though varying in size, are for the most part much smaller than normal. Oc. 5, ob. 9—tube drawn out.—Hartnack.

FIG. 2.—Stage of coagulation of the blood, in which fine fibrillæ, starting into existence over the field, limit, and at last arrest, the movements of the translucent bodies. Oc. 5, ob. 9—tube drawn out.

FIG. 3.—Variations in size of the red blood-corpuscles. Oc. 5, ob. 7—tube drawn out.

FIG. 4.—Aggregation of the translucent bodies into colonies resembling zöoglæa. Plasma of the white elements very diffuent. Oc. 5, ob. 9—tube drawn out.

Fig. 1.

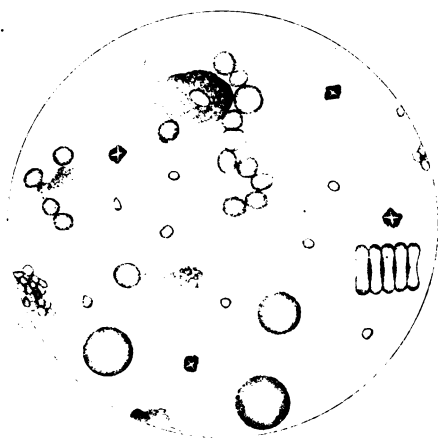


Fig. 2.

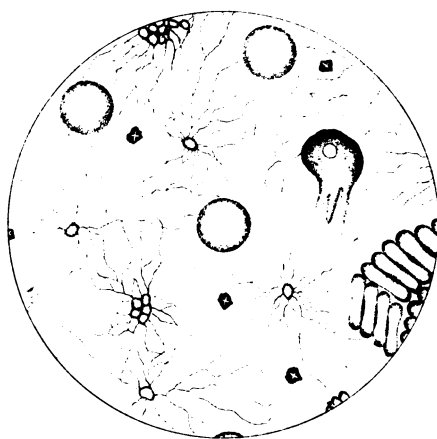


Fig. 3.

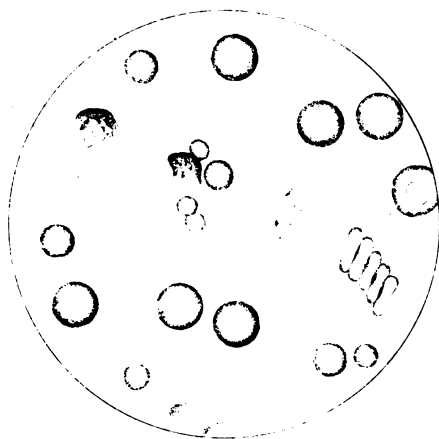
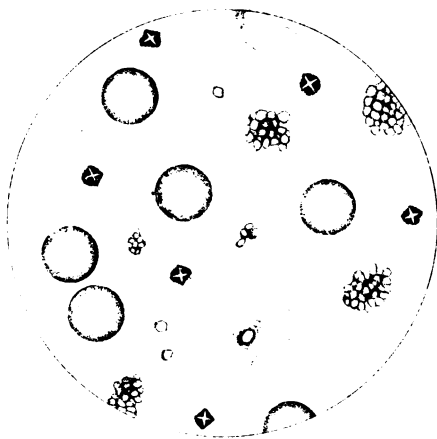


Fig. 4.



ON
A LARGE ANEURISM
OF THE
AORTA, INNOMINATE, SUBCLAVIAN,
AND CAROTID ARTERIES,
TREATED SUCCESSFULLY BY DOUBLE DISTAL
LIGATURE.

BY
RICHARD BARWELL, F.R.C.S.,
SURGEON TO THE CHARING CROSS HOSPITAL.

(Received October 23rd—Read November 13th, 1877.)

ROBERT W—, æt. 45, labourer in a foundry, was admitted into the Charing Cross Hospital, under the care of Dr. Pollock, 24th July, 1877, suffering from aneurism at the root of the neck.

Previous history.—The man has served in the Rifle Brigade in India, and in the Hussars in the Crimea; he has led a hard, rough life, he has had ague, cholera, and dysentery, and has occasionally drunk hard. Although twice affected with gonorrhœa, he appears never to have had syphilis; no scars of chancre or of bubo are detectable. His father and mother are alive and healthy, as are also five sisters and three brothers. There is no family history of heart disease or of aneurism.

Since leaving the army he has worked in a foundry—the

labour chiefly consisting in lifting and carrying heavy weights. With the exception of the above-named acute diseases, he has enjoyed very good health, until November, 1876, when vomiting and purging followed a drinking bout; after that he had pains in all his limbs, back, and shoulders, which were attributed to rheumatism; these all disappeared after a short time, except the pain in the right shoulder. He first noticed a swelling on the right side of the neck in March, 1877; soon after he had occasional swelling on the right side of the head and neck, his right arm became weaker, and was not unfrequently numb. His breath was less good, when he had hard work to do, and he felt his heart beat a good deal.

Present state.—At Dr. Pollock's kind request I examined the man with him. The patient looks more than his age, and has a commencing *arcus senilis*. The aneurismal tumour is situated at the right root of the neck, is oval in shape, with the long axis horizontal above the clavicles; it extends from the left of the middle line from beneath the sternal part of the left sterno-mastoid muscle to near the outer border of the companion muscle of the opposite side; its highest limit is on a level with the upper edge of the cricoid cartilage; its lower boundary, lying behind the clavicle and ribs, cannot be felt. This tumour is pulsatile, with very characteristic expansion, but without any vibratile thrill. Pulsation can be felt in the first intercostal space as low as the border of the second rib, and occasionally through the rib itself for two inches outside the sternum. The radial pulse seems rather weaker on the right than on the left side, but if there be any difference it is hardly perceptible.

Over the tumour no *bruit* is to be discerned, but the two sounds of the heart are readily heard, though muffled; they are audible as heavy dull thuds; the same sounds are also to be heard on the clavicle, first and second rib, and first intercostal space. There is dulness of the upper right chest as far out as the inner third of the clavicle, its outer and lower limit may be traced, in a semicircular direction, from that place over the first intercostal space and second rib.

Internally this area mingles with the cardiac dulness, which extends abnormally far, namely, about an inch and a half to the left, while the apex beats strongly quite half an inch outside the nipple line; probably the heart is also displaced somewhat downward, as there is strong epigastric pulsation. There is no cardiac bruit, but the sounds, more especially the second, are dull and muffled. The heart beats strongly with a heavy thud.

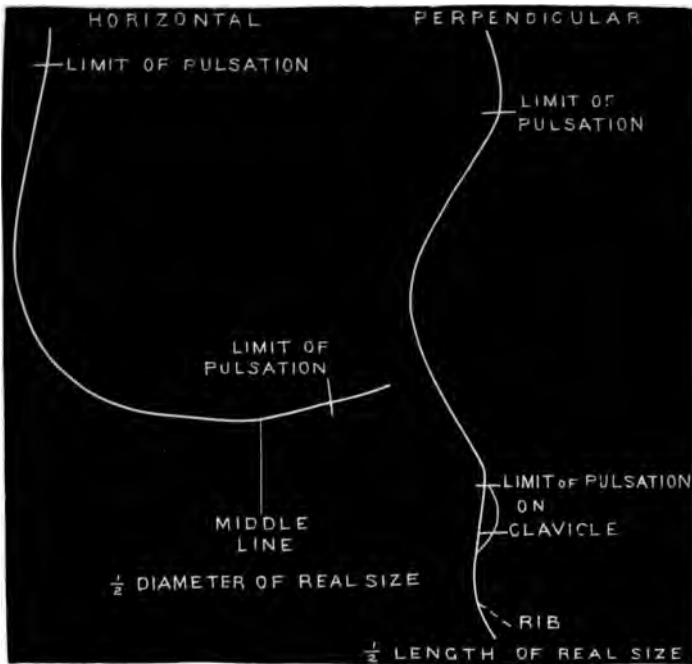
The man has a cough, hardly laryngeal in sound, with copious, diffuent, muco-purulent expectoration. Resonance, except over the above mentioned space, is fair; liquid râles are heard more or less over the whole chest.

The right external jugular vein is distended, as also are the veins of the arm, but in a lesser degree, while those over the sternum and right pectoral muscle are strongly marked. In spite of this affection, the man's constitution seems sound and unbroken; his appetite is good, and he sleeps well if the head and shoulders be considerably raised. The urine, though loaded with lithates, is free from albumen.

Dr. Pollock treated the man with low diet, digitalis, an occasional hypodermic injection of morphia when he was restless, and ice bags to the tumour; but the aneurism increased. Deligation was proposed to him; he accepted readily and at once. On going to the hospital on the following day, I found the man just leaving, and declining any interference. However, I did not lose sight of him, and he, finding the tumour still increase, and reconsidering what I had told him, spontaneously asked me to take him in again and operate. He was readmitted by Dr. Pollock's sanction, under my care on the—

13th August.—In the interval the tumour had increased in size, and somewhat altered in shape, having, like an abscess about to point, become somewhat conical, the blunt apex of the cone lying about two thirds up the tumour, the upper margin of which now reached above the middle of the thyroid cartilage. The pulsation, also, was nearer to the surface, giving the impression of being immediately beneath the skin. The veins of the thorax, upper arm, and the

external jugular were more full and prominent (Plate II, fig. 1). A flexible wire was passed horizontally round the tumour and front of the neck, another perpendicularly from the chest upwards; the tracings thus obtained are subjoined. A compass measurement from the most internal to the most external point of pulsation attained four inches and a half, while tape measurements between the same points amounted



to a little over six inches. From the clavicle to the highest point of pulsation, measured three inches and three quarters by compass, and four inches and a half by tape. These measurements, the wire tracings, and the photographs, furnish all possible means of estimating the size, projection, and appearance of the tumour.

The intra-thoracic portion of the aneurism seemed also

increased as the pectoral dullness extended further to the right, that is, to the middle of the clavicle and to that level in the intercostal space. I consider, also, that the apex of the heart beat still further to the left of its normal position.

On account of this rapid increase in the size of the aneurism, and also on account of the apparent proximity of pulsation to the finger, I judged that there was no time to lose, and I determined to operate on the day after the man's readmission. I felt, however, by no means sure that there was room to place a ligature on the common carotid between the sac and the bifurcation; expecting, indeed, that I might find it necessary to tie both the external and internal trunks.

Operation.—14th August.—Mr. Braine administered ether, and under the carbolic spray, I made a rather smaller incision than usual along the margin of the sterno-mastoid muscle. As soon as the platysma myoides muscle was reached, I became aware of large pulsation inside and close to the carotid artery, occupying an oval shaped space convexity upward. This, I feared, was a prolongation of the aneurism by rupture, in that situation, of all the coats, a new sac being formed merely by condensed surrounding tissues. I therefore felt bound to proceed with the utmost caution, and it was not until a careful dissection of the part showed the cause of the pulsation, that I felt justified in proceeding with greater freedom. This pulsation was produced by a tortuous superior thyroid, so greatly enlarged as to be as big as an external carotid at the level of the lingual. As soon as this condition of the parts was ascertained, I opened the carotid sheath, and found just room enough to pass an aneurism needle below the bifurcation. My first ligature, a catgut one, broke; but a second held, and the isolation of the vessel being verified, was tied, pulsation in the artery above and in the temporal immediately ceased; no difference in the condition of the tumour was observable.

I now proceeded to lay bare the subclavian in its third part; avoiding a greatly enlarged external jugular vein, I reached the vessel rapidly and with remarkably little bleeding and passed an aneurism needle beneath it. Pressure in

the hollow of the instrument caused cessation of the radial pulse. In pressing the needle a little further onward in order to disengage the catgut a gush of venous blood came from the neighbourhood of its point, I brought out one end of the ligature, withdrew the needle and proceeded to search for the bleeding vein. Pressure, backward, above and outside the brachial plexus, commanded the hæmorrhage; but when the finger was withdrawn the cup-shaped wound filled rapidly, rendering it impossible to see the bleeding vein. I therefore thought it unadvisable to make any attempt to tie it or to lose time in ascertaining if the artery alone were included in the ligature, but asked Mr. Godlee to knot the catgut loosely and to leave the ends uncut as a future guide and then with my finger still upon the vein—probably the posterior scapular—stuffed the wound firmly with antiseptic gauze, bandaged it, and allowed the patient to be carried to bed, the radial pulse still beating.

15th.—The patient passed a good night suffering only from occasional tingling in the clavicular wound, to-day he is in excellent condition without fever or pain worthy of notice; pulse 90; temp. 99.2°.

Under the antiseptic spray I removed all the stuffing from the wound, which was dry and fresh-looking, no bleeding or even oozing took place. I traced down with my finger the ends of the ligature and found it round the artery but it also included a branch of the plexus which lay so near the vessel as to seem to the finger in front of it. I determined therefore to leave this loosely tied ligature *in situ*, passed another round the artery, whose isolation was verified and tied the catgut firmly. All this was done without any use of the knife. Although now both radial and carotid pulse were stopped, no change in the condition of the tumour was perceptible, but I observed a marked diminution in the pulsation in the first intercostal space. He was ordered to have No. 1 diet, namely; for breakfast and supper, gruel 1 pint or milk $\frac{1}{2}$ pint, bread 4 oz., butter $\frac{1}{2}$ oz.; dinner, rice milk $\frac{1}{2}$ pint, bread, 4 oz., as an extra, fish.

16th.—Patient in excellent condition, pulse 90, tempera-

ture, morning $96\cdot4^{\circ}$; midday $98\cdot8^{\circ}$; evening $98\cdot2^{\circ}$; he complains of no pain, but on questioning he mentions a dull heavy sensation in the arm. The pulsation over the chest is certainly less and appears to occupy a smaller space.

21st.—Patient's condition is very good, the temperature has never risen above the normal; the pulse varies between 84 and 96; the wounds are healing rapidly without suppuration. Intra-thoracic pulsation greatly decreased, and the apex of the heart is more near to its normal place. The tumour has become firmer, the pulsation less powerful and more distant, it measures by—

Tape	$4\frac{1}{2}$
Compass	$3\frac{1}{2}$

Two eggs are substituted for the fish.

25th.—He is less comfortable and somewhat irritable; temperature still below normal; pulse varying from 76 to 84; tongue rather furred. Intra-thoracic pulsation and area of dulness has decreased a little since last report; action of the heart less violent; epigastric pulsation has nearly disappeared. The tumour, however, remains of the same size and the pulsation appears to my finger rather nearer. As this progress is not so rapid as it was at first I determined to try a very dry diet, and ordered for breakfast and supper, bread 4 oz., butter $\frac{1}{2}$ oz., milk 2 oz.; dinner, meat 3 oz., bread 3 oz., water 4 oz.

26th.—Slight pulsation, evidently collateral in the radial artery.

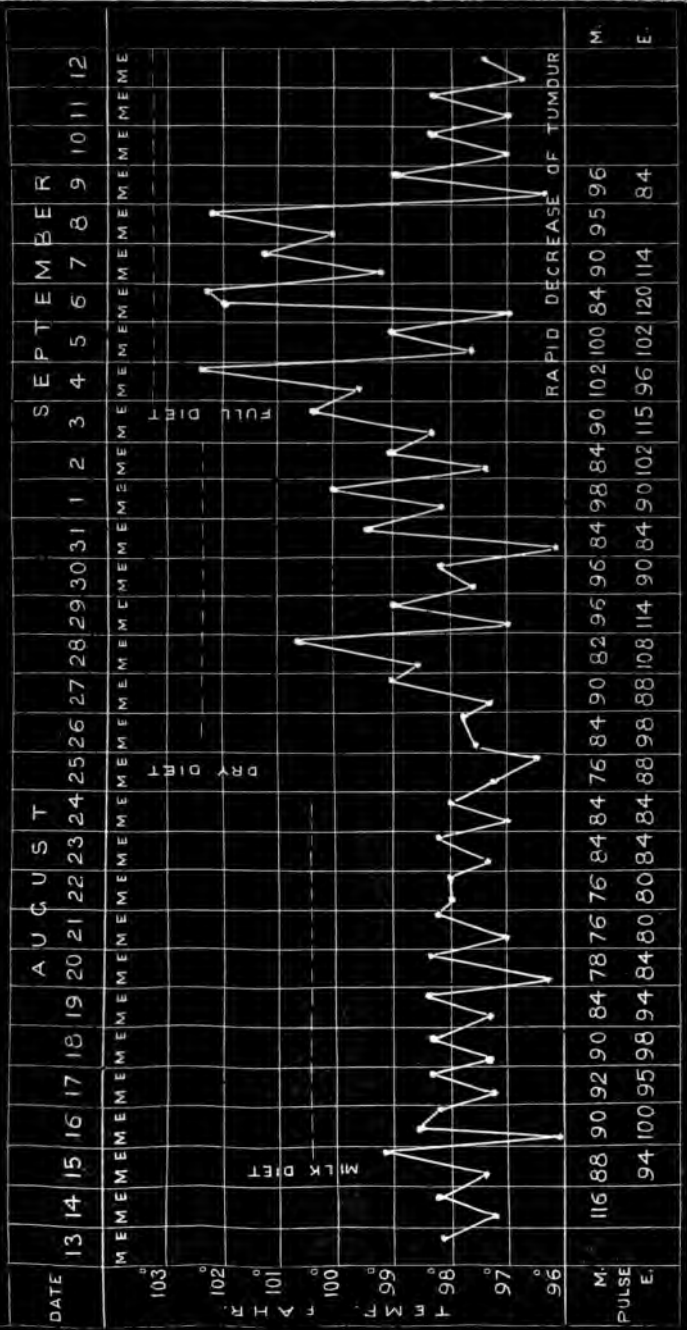
28th.—The patient is much annoyed by the restrictions of his diet table, and his thirst is considerable. I have allowed him to take a little more fluid; but the desirability of continuing the starving system is doubtful; the man rebels against it, his heart-action is excited, and his temperature has become irregular.

The pulsation in the radial has ceased again, that in the tumour is increased, and that on the thoracic parietes is also more perceptible, although the area still seems to diminish. As the pulse was 102 I ordered him to have 15

NAME
ROBERT.W

AGE
45.

DISEASE
ANEURISM OF AORTA



minims of tincture of digitalis every six hours, the dose to be increased until the pulse became somewhat reduced.

30th. The heart's action is still quick, from 96 in the morning to 114 in the evening. The temperature is very variable, the highest has been $100\cdot6^{\circ}$ the lowest 97° , namely on the evening of the 28th, and morning of the 29th respectively. As the digitalis nauseates without diminishing the pulse it is to be discontinued. Measurement of tumour by

Tape	$5\frac{1}{2}$
Compass	$3\frac{1}{2}$

an increase in breadth of $\frac{1}{2}$ inch, but no increase of projection.

September 3rd.—The patient's temperature and pulse remain in much the same condition; the skin is rather dry and hot; the man appears in an irritable state. The heart beats much nearer its normal place and its impulse is violent. The area of dulness in the right chest has somewhat declined, but the visible tumour is much the same in size, and the pulsation seems as near. I changed the diet to No. 3, namely, for breakfast and supper, gruel 1 pint, milk $\frac{1}{2}$ pint, bread 4 oz., butter $\frac{1}{2}$ oz. As an extra at breakfast 1 egg. Dinner, meat (cooked) 4 oz., bread 4 oz., potatoes $\frac{1}{2}$ lb., he also had beer, 1 pint.

I also prescribed of Liquor Ferri Perchloridi 12 minims, and of dilute Hydrocyanic Acid 3 minims every six hours.

5th.—Yesterday the patient seemed better although the temperature rose in the evening to $100\cdot2^{\circ}$. The pulsation has receded and the tumour is smaller. Measurement—

Tape	$4\frac{1}{2}$
Compass	$3\frac{1}{2}$

8th.—

Tape	$3\frac{1}{2}$
Compass	$2\frac{1}{2}$

9th.—

Tape	$2\frac{1}{2}$
Compass	$2\frac{1}{2}$

10th.—

Tape	$2\frac{1}{2}$
Compass	$2\frac{1}{2}$

These measurements are given consecutively that a succinct view of the decrease in size of the tumour may be obtained. During the same time in the upper part of the chest, the area of dulness and of pulsation, whose diminution had up to the 28th been more regular than that of the visible tumour, and in which no period of increase could be detected, rapidly became smaller, and respiratory murmurs were taking the place of the dull thuds over the greater part of the space; cough had almost disappeared, as also the liquid *râles*.

There remains to remark that, on the evenings of the 4th, 6th, and 8th, the temperature rose up to $102\cdot4^{\circ}$. After that, viz., on the 9th, it went down to $96\cdot4^{\circ}$, and has since remained normal. On the 9th he complained of peculiar hot tingling pain from the shoulder to the hand. The limb is not swollen, but the skin chiefly along the course of the vessels is marked with interrupted broad pink lines. These are certainly not due to phlebitis, but I believe to an inflammatory condition of the empty arteries, there being no pulse in any of the brachial vessels. I ordered stimulation of the skin with a diluted ammonia liniment; and as the pain interrupted his usually excellent sleep, a small dose of the *Liquor Opii Sedativus*. The pain and redness disappeared in two days, and the opiate was discontinued. A slight enlargement, more perceptible to touch than to sight, remained. Measurement by

Tape	$1\frac{1}{2}$
Compass	$1\frac{1}{2}$

On the 11th I left town, and the case fell under the care of my friend, Mr. Bellamy. I find noted, on the 13th, the tumour is about the size of an average walnut, very hard on its inner side, and above the sterno-clavicular joint; it still, however, pulsates.

23rd.—Axillary and brachial arteries are swollen, hard, and excessively tender; their enlargement is even visible; they were painted with iodine.

30th.—The pain and swelling mentioned in last report disappeared in three days; the tumour has still further diminished.

October 1st.—On my return I found the man apparently in very good health, and without any visible enlargement at the root of the neck. On passing the finger behind either edge of the sterno-mastoid muscle, and pressing gently downward, one feels a tumour about the size of a pigeon's egg, which, although quite hard, pulsates; it is doubtful if the pulsation be at all expansile, or if it be not simply communicated by the aorta. I myself consider it somewhat expansile. There is neither radial nor ulnar pulse; nor, indeed, any arterial pulsation in brachial or axillary artery, yet the limb is warm and well nourished. A feeble pulsation may generally, but not always, be felt in the temporal.

The condition of the chest, as kindly furnished me by Dr. Pollock, is as follows:—There remains a slight cough, but not, the man says, as much as he has had ever since he returned from the Crimea; the mucous *râles* have disappeared. Over the right side of the manubrium sterni, and about half an inch outside it, there is some dulness, and over this, and a little further out, the heart-sounds, more especially the second sound, are too distinctly heard, and with a slight ring: no pulsation is to be felt at this part. The heart has returned to its proper place, its beat is of normal force; the second sound is somewhat dull and muffled, in all probability from thickening of the aortic valves.

Dr. Green, I may remark, thought the physical signs indicated contraction of the lumen of the aorta rather than increase.

The man was taking no medicine, but has been put on No. 4 diet, and was given one pint and a half of beer daily, this I diminished to one pint. No. 4 diet consists of the same as No. 3, but with six ounces instead of four ounces of meat at dinner.

On 12th October I had the man photographed; by an error he was allowed to get out of bed and sit up for two hours, walk the length of the ward some twenty-five yards, and undergo the fatigue of sitting for the likeness. When I saw him the pulsation seemed more expansile—the heart's action

was considerable. This condition led me at that time and during the few following days to investigate the condition with even more care; I believe the sense of an expansile pulsation to have been deceptive.

At this date of writing, 20th, the tumour in the neck rises only just above the sterno-clavicular joint, and can only be felt by passing the fingers behind the sterno-mastoid; it is rather conical in shape, and the impulse communicated from the aorta pushes the cone upward between the fingers like a wedge, and by thus separating them gives a false idea of expansile pulsation; by taking the tumour a little firmly between finger and thumb, and lifting it ever so slightly, pulsation ceases. Nevertheless, I deemed it the better plan to keep the man in bed until the 22nd, although I believe the whole aneurism to have become filled with firm clot, and that the innominate, carotid, and subclavian arteries are entirely obliterated.

REMARKS.

The notes of this case have occupied so much time, since a somewhat detailed report appeared to me desirable, that the few remarks I have to make shall be compressed into the smallest possible space.

Nature of the aneurism.—The generally healthy condition of the man's circulatory system, and the history of work involving frequent and violent efforts, lead to the belief that the aneurism was traumatic in origin; produced, that is to say, by some great exertion when the system was depressed by a period of hard drinking.

The shape and position of the tumour show clearly that the innominate, together with its two great branches, was involved in the aneurism, moreover, the pulsation felt in the first intercostal space, even occasionally through the second rib, and the extent of pectoral dulness point strongly to the conclusion that the aorta also was implicated. It was impossible to trace by percussion the cardiac side of the tumour,

but the very considerable displacement of the heart, namely, from one inch and a half to two inches to the left, would show that the intra-thoracic portion of the aneurism must have been very large. I know perfectly the difficulties which surround clear diagnosis of these conditions, but I can entertain no shadow of doubt that the first part of the transverse aorta, the innominate, nearly all the common carotid, and at least, the first part of the subclavian arteries had opened out into a large blood-filled cavity, whose dimensions have been given with all obtainable precision.

Operation.—Of the operation I would only remark that although the unfortunate rupture of a vein caused me to forego for twenty-four hours the definitive deligation of the subclavian, yet the tying of the two vessels must be ranked as simultaneous.

Diet.—The regimen, upon which my patient was at first put, resembled very closely that on which a patient of Mr. Stanley's, with large aortic aneurism, recovered.¹ On the tenth day, although the relief to the circulation was progressive, as the diminution of the tumour seemed at a standstill, a very dry diet was prescribed. On the third day of this system, the heart and arteries generally took on an excited action, and the temperature became disturbed; nevertheless, the same regimen was continued for nine days, when a fairly nutritious diet with a little stimulus was substituted, yet the excited heart-action and irregular temperature continued for five days after this change.

The relation between diet, temperature, and decrease of the tumour.—During the first five days of dry diet and excited heart-action, the tumour increased half an inch in breadth, it then remained of the same size for four days. Two days after the administration of a fuller diet and a little beer, that part of the tumour which was situated in the neck, began rapidly to diminish. The conduct of the intra-thoracic portion could be less easily verified, it appeared to me that during the phase of vascular excitement its decrease was rather slower.

¹ 'Pathological Transactions,' vol. v, p. 107.

It is to be expected that some divergence of opinion will prevail as to the effect of the whole diet system upon the disease. Some will see in the vascular excitement only a parallel to that phase of increased pulsation, of pain, and of heightened temperature, which, when an aneurism is treated by pressure, often precedes the successful issue.¹ Others may consider that the dry diet abetted, perhaps actually induced an inflammation of the sac, eventuating in coagulation of its contents. Such view would certainly be supported by the occurrence of arteritis in the arm four days after the tumour began to diminish. Again, it may be considered that the diet system had produced in the blood, as I had intended it to do, a highly coagulative tendency. In confirmation of this view it should be remarked, that the inequality of temperature corresponds closely in time with the cessation of radial pulse after its reappearance for two days. Since no pulse can even now (Oct. 21th) be felt in the radial, we must conclude, that the whole subclavian artery became occluded with blood-clot, for it is hardly conceivable that radial and ulnar, brachial, and even axillary pulsation, should remain absent if the first part of the subclavian, with its large scapular branches were patent.

It seems certain that the systemic symptoms noted between the 28th August and 8th September were closely connected with the changes, first increase and then decrease of the sac; also, it seems certain that these symptoms were closely connected with the diet.

Result and Records.—At the time I operated the tumour in the neck was so large, its walls so thin, the circulation and breathing so embarrassed, and the heart so displaced, that a fatal issue, but for the steps taken, was closely impending. Nevertheless, in the absence of any distinctive evidence of general arterial disease, I conceived the case to be a hopeful one. This view was justified by that relief

¹ A description by an intelligent gentleman of his sensations during the last few hours of treatment by pressure, and the sudden cessation of uneasiness when the aneurism became almost suddenly consolidated, was read by me to the Clinical Society, and is published in the 'Transactions' (vol. v, p. 110).

to the breathing and to the circulation which almost immediately followed the operation, showing, even though the cervical part of the tumour was unaltered, that some considerable change of the thoracic portion had occurred. Nevertheless, in speaking of any such case as hopeful I do so with distinct reference to past records. Since the days of Wardrop it has been theoretically held by surgeons that "innominate aneurism is or should be amenable to treatment by ligature of the carotid and subclavian." Mr. Holmes, in his lectures on surgery, gives six cases of the simultaneous deligation of the carotid and third part of the subclavian; from these one must be eliminated, Mr. Heath's, since in his case the aneurism proved to be aortic only. One more case, however, Mr. Ensor's,¹ must be added. Of these six cases one received no benefit, but lived some time; two, who also were not relieved, died in a few weeks; one (Mr. Ensor's) received hardly any, if any, benefit, and lived sixty-five days; two died on the sixth day. My case is then the only one which, having been successful, proves practically the above theory to be correct.

As far as I know, my case is also the only one which has been operated on with all the antiseptic precautions duly observed; and I consider the temperature chart (until the diet was changed) a remarkable fact and one well worthy of notice. Different opinions will doubtless be entertained as to the influence which this method and the absence of morbid temperature may have had on the result. My faith in this absence of wound-fever and in the value of carbolized catgut is very great.

Deductions.—It seems impossible to avoid the following conclusions:—That not only innominate aneurism, but such aneurism when the aorta is largely implicated, is amenable to treatment by the double distal ligature. Moreover, we may conclude—what, indeed, was proved by Mr. Heath's well-known case—that aneurism of the first part of the aorta may be cured by the same procedure. It is true that deligation of the first part of the subclavian would be the more logical

¹ 'Lancet,' 1875, vol. i, Feb. 15, and vol. ii, July 31st.

operation, but it would also, I believe, prove the more deadly one.

SEQUEL.

Robert W—, the subject of the above communication, was exhibited to the Fellows of this Society when the paper was read, 13th November, 1877. He left the hospital on the following day.

On the 20th November he returned to the hospital to present himself at the weekly board, and I, finding him suffering from bronchitis, persuaded him to come in again under the care of Dr. Pollock. At that time the cervical portion of the tumour was no larger than when he was shown in these rooms, perhaps even a little smaller. He had, however, bronchitis over the whole chest and some pneumonia at the back of each lung. He was very morose and indocile, demanding other food and drink than had been prescribed for him, and when on the 22nd the house-physician ordered the discontinuance of a certain allowance of brandy he left the hospital in a rage, although his dangerous condition was fully explained to him. The weather just then was very cold and damp, sleet was falling, and the man was very insufficiently clad. I learnt afterwards that when he reached home his wife was away, there was no fire, and he sat in his thin wet garments in a perfectly cold room for more than four hours. When his wife returned she found him pale, almost blue, and breathing with great difficulty; he did not appear quite conscious. He continued throughout the short remnant of his life to get worse in these particulars.

24th.—Mr. Colquhoun, a late pupil of the hospital, was seeing patients for a friend at a small dispensary in Brompton, when a woman came in and begged him to go and see a man dying close by. He went and recognised my patient W—, moribund, apparently from pneumonic asphyxia; he died in a few minutes. Mr. Colquhoun at once made me

acquainted with the occurrence, and I was able to arrange for a post-mortem examination on the following day.

25th. *Post mortem*, twenty-three hours after death.—The body was well nourished, there was considerable rigor mortis and posterior decubital staining. The tumour just above the right sterno-clavicular joint felt, except, of course, that it did not pulsate, exactly as during the last few weeks of life.

The chest was opened by a crucial incision from the thyroid to the ensiform cartilage, and from one acromion to the other, the ribs being sawn through a considerable distance from the junction with their cartilages, and the clavicles disarticulated. On removing the front wall of the thorax the presence of an aneurismal tumour was manifest. The pericardium was opened, it contained about five ounces of slightly turbid serum. The heart and great vessels were removed entire without further examination. On dividing the left subclavian artery a large quantity of blood, as black and thick as post-mortem venous blood, escaped from its proximal end. Nothing further was remarked at this time except that all the veins were full.

The lungs were markedly œdematous, a quantity of clear but æriferous serum bubbling forth upon pressure; there was also a good deal of hypostatic pneumonia; the portions of lung so affected felt solid, but broke down readily; there were no cavities, no new deposit, or pathologic breaking down of tissue. The bronchi, even to very minute divisions, were strongly congested and filled with mucus or with mucopus.

Brain—healthy, firm, but congested; veins of the pia mater, choroid plexus, &c., very full of dark blood.

Abdominal organs healthy.

The parts removed were taken at once to the hospital and preserved in spirit for subsequent examination.

In the examination of this tumour I was assisted by my friend Mr. Cantlie.

The trachea and œsophagus were pressed by the tumour to the left side, the former being somewhat flattened from the

right, but not enough to cause any obstruction to the entrance of air; it was also pressed backward, so as to lie in a plane a little posterior to the carotids. Moreover, these tubes were twisted, so that about the level of the top of the sternum, where the twist is greatest, the œsophagus lay to the left and a little in front of the trachea, whose non-cartilaginous portion looked wholly to the left.

The aneurismal tumour lay to the right of and above the first part of the aorta; looked at from the front it was seen to extend above the first bend of that vessel, covering the lower part of the carotid and subclavian arteries. Lying against its left side was the left vagus nerve, slightly flattened, and, perhaps, a little deflected. Winding round its lower part was the left innominate vein, elongated and bound to the wall of the tumour by condensed fibrous tissue.

The veins of the right side were peculiar; the subclavian joined, about an inch from the heart, a large venous trunk formed by the right internal jugular with thyroid axis branches, and by the left innominate. Thus, the right subclavian appeared to enter almost at once into the auricle. Whether this was an original peculiarity or a modification by disease I cannot say with certainty, but believe that it was the former, since in this case the right subclavian entered the larger venous trunk on a level considerably lower than that of the left innominate.

The aneurism sprang from the junction of the first and second part of the aorta; extending upwards and to the right, it involved the front wall of the innominate together with the roots of the right carotid and subclavian. It was rather larger than a tennis ball and had a subsidiary projection upwards and outwards, which was divided from the bulk of the tumour by a broad shallow groove, probably the impress of the clavicle. The back part was moulded on the apex of the lung. In texture the whole mass, after a certain immersion in spirit, was as hard and unyielding as a solid piece of leather (see Pl. II*).

The whole tumour was cut through from before backward; it was then seen that a globular cavity, communicating freely

with the aorta, still existed. This was perhaps a little more than an inch in diameter; it was surrounded by very firmly laminated clot of variable thickness. In front, the thinnest part, it measured over a third of an inch. At the back it was an inch and a half thick. The subsidiary part was entirely filled up, constituting a mass of firm fibrin about two inches in thickness. The walls of the aneurism on the section surface were distinguishable.

Below the mouth of the cavity, therefore, on the wall of the aorta itself, the openings of the left carotid and subclavian were seen to be rendered somewhat oblique and elliptical by pressure.

The innominate, subclavian and carotid were obliterated, firmly plugged by coagulum, which on the section of one of them had a pink colour like the lean of ham.¹ On the subclavian the remnants of two ligatures might be seen like delicate shreds of connective tissue; there was no wound or scar on the outer coat of the vessel. The same may be said of the right carotid, except that the trace of only one ligature of course was found.

Remarks.—In considering this case in the light of the conditions disclosed by anatomical examination I must, while drawing attention to the general accuracy of the diagnosis, point out that the aneurism did not involve the carotid to the extent which it seemed to do during life. The part of the tumour which I have, in the description of dissection, called subsidiary, and which is so strongly marked in the the first photograph, surged up from lower parts and covered rather than involved any large part of the cervical vessel. The mode by which I accounted for the deceptive sense of pulsatile dilatation in the cervical portion of the tumour during the latter part of the man's stay in the hospital is remarkably borne out by the dissection.

The position of the trachea and œsophagus, together with

¹ By some misfortune, in the very dark room where the post-mortem was made, the subclavian was cut through about the beginning of the second stage, but the rest of the vessel still attached was brought away, and the accident was not discovered till afterwards.

the almost spiral course of the left innominate vein round nearly to the back of the tumour, show that also the thoracic part of the aneurism must have been very much larger before operation than for several weeks previous to death.

Lastly, I may direct attention to the large quantity of firm organized fibrin deposited in the aneurism, to the comparatively small size of the cavity still existing, and to the fact that as no vessel leads out of it there could not be any stream of blood within it. It will therefore be conceded me that had this patient been of more tractable temper, had he either remained in hospital or been moderately prudent out of hospital, the whole tumour, since it had no thoroughfare, and since such strong walls could neither dilate nor burst, would have become, of necessity, filled up. In fact, that although the man died of an intercurrent disease brought on by his own obdurate indocility, his aneurism was to all intents and purposes cured.

DESCRIPTION OF PLATES II & II*.

Aneurism of Aorta, &c. (Richard Barwell).

PLATE II.

FIG. 1. Robert W—, from a photograph taken immediately before the application of the double distal ligature.

FIG. 2. Robert W—, from a photograph taken seven weeks after the operation.

PLATE II*.

Robert W—, Dissection of the aneurism, arch of the aorta, large vessels, and neighbouring parts.

Fig. *a*. Aorta.

b. Aneurism nearly filled.

c. Left carotid artery.

d. Left subclavian vein.

e. Left vagus nerve.

Fig. *f*. Left innominate vein.

g. Trachea

h. Œsophagus } twisted.

i. Right carotid artery.

k. Right jugular vein.



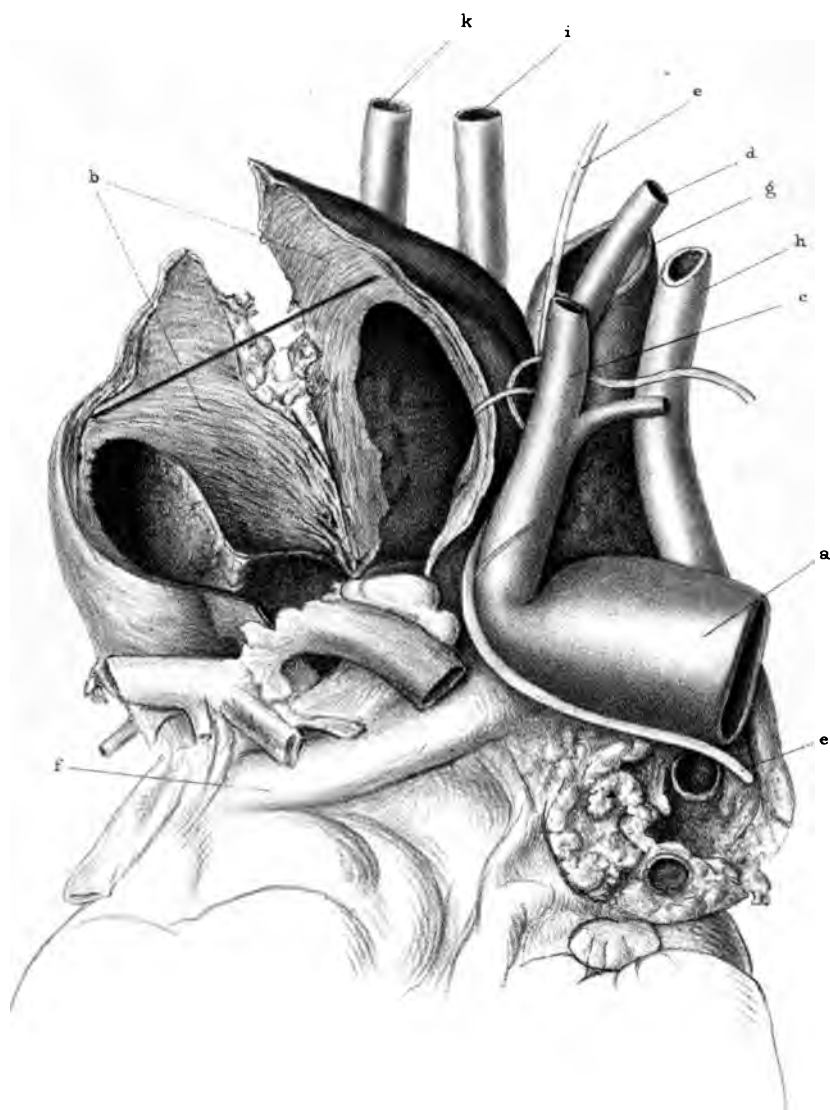
Fig. 1.

R. Mintern lith.



Fig. 2.

Mintern Bros imp.



ON THE
REMOVAL BY OPERATION
OF A
HAIRY MOLE OCCUPYING HALF THE
FOREHEAD.

BY
W. MORRANT BAKER, F.R.C.S.,
LECTURER ON PHYSIOLOGY AND ASSISTANT SURGEON TO ST. BARTHOLOMEW'S
HOSPITAL, AND SENIOR SURGEON TO THE EVELINA HOSPITAL
FOR SICK CHILDREN.

(Received November 13th—Read November 27th, 1877.)

THE fact that surgeons not unfrequently decline to treat by operation large hairy moles, fearing that the disfigurement produced either by the knife or caustics might be worse than the disease, will, I trust, seem a sufficient excuse for bringing the present brief communication before the Society.

The patient, whose case forms the text of this paper, (M. G—), ten years old, was admitted into St. Bartholomew's Hospital under my care, in February 1873, on account of a large congenital hairy mole on the forehead.

The mole occupied rather more than half the forehead, extending from the hairy scalp to the eyebrow, and from the temple to the middle line, and a little beyond it. Its greatest diameter (from side to side) was three inches and a half, and its vertical diameter two inches and three quarters. The integument composing it was deeply pigmented, being of a

dark, blackish-brown tint, with a very uneven surface, especially below and to the left, where it was mammillated or almost warty. A quantity of brown coarse hairs grew from nearly every part of the surface, being thickly planted, as in the case of the hairy scalp, and many of them an inch or two in length. In the centre of the patch was a small area of almost white skin, like scar-tissue, the hairs growing from which were also of a lighter tint than the rest.

The mole was vascular, and, it was said, always bled profusely when in any way hurt; but it had only grown in proportion to the rest of the body. (See Plate III, fig. 1.)

The child was in good health, and well grown for her age, and, excepting the almost startling deformity produced by the mole, good looking.

I was unable to find any account of the successful employment of measures for the removal of so large a mole, and the treatment to be adopted could be, therefore, only experimental. It appeared to me, however, that with care, it would be impossible to make matters worse, and an attempt to improve them would be quite justifiable.

A few days after the patient's admission into the hospital I began the treatment, after the patient was put under the influence of chloroform, by cutting off a small portion of the surface of the mole as smoothly as possible with a sharp scalpel; taking care, while cutting away both the epidermis and corium, not to remove the whole thickness of the latter, in order that the subcutaneous tissue might be left untouched, and thus such contraction as is apt to follow its destruction might be prevented. Free but not excessive bleeding occurred from every part of the cut surface. It was controlled easily by a pad and bandage.

Two days afterwards the dressing was removed and the surface allowed to form a scab, which was not touched for several days.

March 21st (a month after the operation).—The wound had healed with a good scar, which was thin and red, but not pigmented. Some new hairs are beginning to grow from the surface, but not so thickly as before, especially at the

spots at which the knife had gone most deeply. There was no contraction.

March 26th.—Encouraged by the result of the first operation, and especially by the total absence of any contraction of the scar, which was as smooth as the neighbouring healthy skin, I now attacked the remainder of the mole with the knife, cutting away the whole of its surface, including, as before, both the cuticle and part of the cutis vera, but not exposing, except here and there to a very small extent, the subcutaneous tissue. The bleeding was controlled by a pad of lint and a bandage, and after a day or so the wound was left to heal by itself under a scab, such discharge as there was being allowed to escape by occasionally raising the edge of the scab.

In less than a month after the second operation the wound was almost completely healed. The scar was smooth and not pigmented, but hairs had begun to crop up again pretty freely from its surface.

So far as it went the result of these operations was satisfactory, especially in regard to the proof they afforded that large portions of skin could be removed without consequent contraction and deformity of neighbouring parts. But it was evident that the skin must be destroyed to a greater depth in order to get rid of the larger hair-follicles.

May 23rd.—Nitric acid was applied to a small portion of the surface, the scab produced by the acid being left untouched, and little or no dressing of any kind applied.

Nitric acid was again applied to another portion June 28th. At this date it was noted that there was a slight reappearance of brown pigmentation near the lower border of the scar.

The caustic was again applied freely to the upper portion of the mole August 29th, and a few days afterwards the patient was discharged from the hospital for a few weeks.

On her readmission, October 1st, the upper part of the mole was found still covered by the scab produced by the nitric acid. The lower part was pinkish and smooth, with a considerable number of fine hairs still growing from it.

In December the scab, which had been left to itself almost

entirely, had fallen off, and the scarred surface was now pink and glazed. In some parts fine hairs were still springing up. There was not the slightest contraction.

Dec. 28th.—Several spots, from which fine hairs were growing, were touched with nitric acid, and the patient was discharged from the hospital with a promise of readmission in a few months.

June 29th, 1874.—At this date the patient was readmitted into the hospital. Her appearance was very much improved. Very few hairs, comparatively, now grew from any part of the scarred surface, and those present were of much smaller size than their predecessors, and of a lighter colour.

The scar had become more like normal skin, although still looking glazed.

Glacial acetic acid was applied to some of the spots at which hair was still growing (July 6th), but the remedy seemed scarcely strong enough, and nitric acid was applied July 21st.

September 22nd.—The scabs had fallen off at this date from the lower part of the forehead, leaving smooth scar-tissue, thin and supple, and with only a few hairs growing from the surface.

The patient left the hospital soon afterwards, and was not readmitted until—

February, 1876.—At this time a marked improvement was perceptible. The greater part of the disease had quite disappeared. The scar was smooth and supple, and there was not the slightest contraction or dragging on neighbouring parts. A few small pigmented and slightly hairy spots were still present here and there.

Several of these spots were treated by epilation and the application of nitric acid, a drop of the acid being applied, and, after standing for a few moments, being soaked off with blotting-paper. This was done on two or three occasions, and the patient was discharged June 11th.

On her readmission at the beginning of last month (October, 1877), it was evident that the mole had almost entirely disappeared, the only traces of its presence being a tiny patch

here and there of slightly pigmented skin, with a very few fine hairs, almost imperceptible except on close inspection, growing from them.

The scar-tissue is now smooth, whitish, glazed and supple, quite level with the adjoining healthy integument, and not in the least degree contracted, or dragging in the least on neighbouring parts.

Some of the hairs I have attempted to remove with their papillæ, by means of the point of a small tenotomy knife, on one or two occasions; but with this exception, and the removal of the small slice of skin just at the edge of the hairy scalp, nothing has been done since her readmission.

A photograph, taken in the last week of October, shows well the slight amount of disfigurement which now remains (see Plate III, fig. 2). No photograph was taken before the beginning of the treatment, but a photograph of the drawing which was made at this time is placed, for comparison, by the side of the photograph recently taken from life (see Plate III, fig. 1).

So far as I can discover, this is the largest hairy mole which has been completely removed by operation.

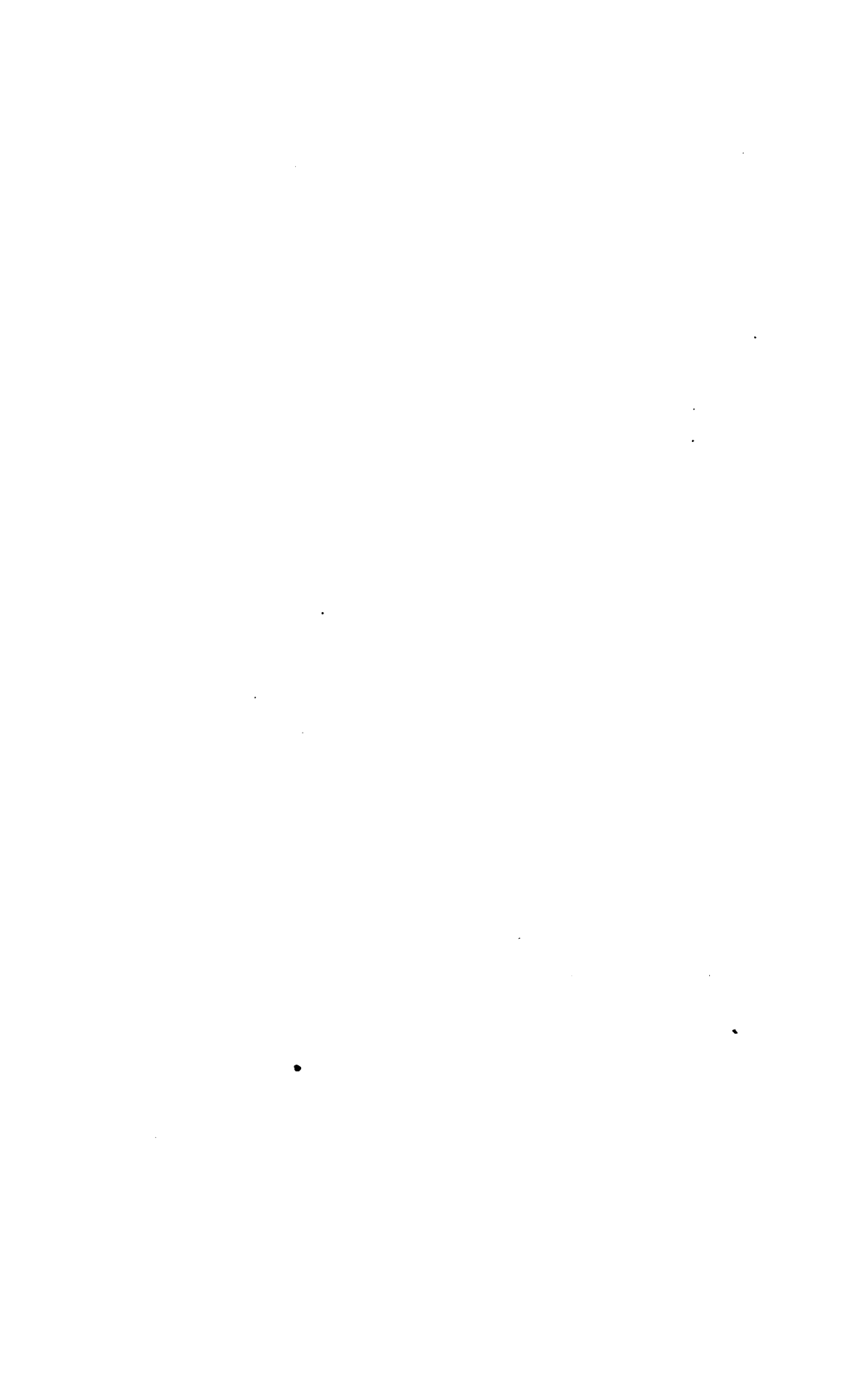
The point of most surgical importance seems to lie in the fact that so large a piece of integument as half that of the forehead can be removed without contraction of neighbouring parts. It is, of course, easy to be wise after the event; but to most surgeons such an entire absence of contraction as there is in the present case will be, I venture to think, as much a matter of surprise as it was to myself. It is impossible to say to what depth the destructive action of the caustic extended, or, in other words, whether the whole thickness of the cutis was destroyed, or what amount, in addition, of the subcutaneous tissue. It is possible that the hair-producing power of the hair-papillæ and their follicles may have been destroyed by something short of complete removal of their whole texture; for in the absence of a supposition of this kind it is not easy to understand how the growth of hair could have been prevented, except by such destruction of the subcutaneous tissue in which the larger

hair-follicles are seated as must have led to contraction when the wound was healed.

With regard to the methods of treatment adopted in this particular case, there must be also some doubt as to the relative amount of good done by the knife and the caustic. The result of the first operations (with the knife) quite confirmed the idea which led me to perform them, viz. that a large portion of skin may be cut away without fear, if the incisions are confined, or nearly so, to the cutis vera; and the hint may prove useful in cases in which the disease is superficial enough to be thus removed. In the present case, however, the pigment-forming tendency inherited by the new cuticle was too strong to be thus eradicated completely, and the larger hair-papillæ were, of course, too deeply seated. It is quite possible that a repetition of the operation by cutting, without any application of caustic, would have sufficed in the end for the cure of the disease; but this must remain for the present doubtful. My object in changing the method from the knife to caustic was partly to experiment with one as against the other, and partly because it seemed not unlikely that the liquid caustic might find its way into the hair-follicles, and thus affect, to a slightly deeper level than the general surface of the integument, those structures which one wished more particularly to attack.

I am disposed to attribute some part, at least, of the evenness of surface and absence of contraction of the scar to the fact that the scab was allowed to remain untouched until the wound beneath was soundly healed.

The process of healing by what may be termed the natural method—by scabbing, is less often adopted, I am disposed to believe, than it should be; and the result of this method in other superficial wounds than those now the subject of inquiry, as, for example, in slight burns or like injuries, confirms me in this belief.



DESCRIPTION OF PLATE III.

Removal by Operation of a Hairy Mole (W. Marrant Baker).

Fig. 1. Photograph from a drawing taken in 1872.

Fig. 2. Photograph from life (1877) after removal.



Fig. 1.

R. Mintern lith.



Fig. 2.

Mintern Bros imp.

CASES
OF
BRANCHIAL FISTULÆ IN THE
EXTERNAL EARS.

BY
SIR JAMES PAGET, BART., D.C.L., LL.D., F.R.S.,

Received November 18th—Read November 27th, 1877.

BRANCHIAL fistulæ in the neck have been often and fully described in Germany, but have been so rarely noticed in this country that some short account of them may be useful for the better understanding of the rarer branchial fistulæ in the ears which it is my chief purpose to describe. They were first observed by Dzondi in 1829, and he gave them the name of Congenital Fistulæ of the Trachea, in the most probably erroneous belief that they opened into that canal.¹

Ascherson, in 1832, besides adding many cases to those by

¹ See Heusinger, 'Virchow's Archiv für Pathol. Anat.,' xxix, p. 365, for explanation of Dzondi's probable mistake. No communication with the trachea has been suspected in any cases since Dzondi's, but three or four cases are recorded of congenital tracheal fistulæ in the middle line of the neck. These were certainly not branchial fistulæ; they may have been due to abscess opening into the trachea and through the skin, or may have been due in part to defective median closure of the branchial arches, after the manner of the malformation in ectopia cordis or ectopia vesicæ. See Fischer in v. Pitha u. Billroth's 'Handbuch,' B. iii, Ab. 1, L. 3, S. 41.

Dzondi observed the communication of some of the fistulæ with the cavity of the pharynx, and explained them as due to incomplete closure of the branchial clefts or fissures of the early foetal state. Several more recent writers, especially Heusinger,¹ have confirmed and extended Ascherson's descriptions and their interpretation; and I have found them true in three instances observed in the last three years.

In general terms, the cervical branchial fistulæ appear congenitally as very fine canals opening into minute orifices in one or both sides of the fore part of the neck, and leading backwards and inwards, or backwards and upwards, towards the pharynx or œsophagus. In some instances a single fistula is found; in others two; in a few three. In the greater number the orifices are just above the sterno-clavicular articulation; in others they are near the level of the upper border of the thyroid cartilage at the anterior edge of the sterno-mastoid muscle. When two orifices are found they are usually placed symmetrically; when three, it is usual for two to be placed at the lower level and one at the upper. In length the fistulæ are various, from half an inch to two and a half. In diameter they vary from that of an ordinary bristle to that of an ordinary probe, the orifices being usually smaller than the canals and less easy to dilate. At their distal ends the fistulæ are most frequently closed, after the manner of blind or incomplete fistulæ; in only a few instances they have been found complete, opening into the pharynx or the upper part of the œsophagus, but by apertures so narrow as only to admit minute probes, or the forcible injection of fluid. The lining of the fistulæ is a smooth, shining membrane, commonly secreting a clear mucous fluid; and, in one of the cases which I have seen,

¹ Loc. cit., and in the same 'Archiv,' B. xxxii. Translations of the papers of Dzondi and Ascherson are published in Clay's 'British Record of Obstetric Medicine,' vol. i, 1848. Heusinger gives references to about forty cases published before 1866; and references to later cases are given by Fischer, loc. cit., and by K. F. v. Heusinger in the 'Deutsche Zeitschrift für Thiermedizin,' B. ii (as quoted in 'Virchow and Hirsch's Jahresbericht' für 1867, p. 227), gives a complete account of all forms of cervical branchial fistulæ in domestic animals as well as in man.

this secretion was always augmented during any accidental bronchial or nasal catarrh. Very rarely portions of cartilage, such as may be thought arrested rudiments of cervical ribs, have been felt under the skin near the openings of the fistulæ.¹

The cervical branchial fistulæ appear, as a rule, to undergo no material change from birth to the end of life. They may become so troublesome by their secretion as to need surgical treatment; and I believe that the best treatment is with the galvanic cautery applied through their whole length.

Such are, generally, the cervical branchial fistulæ, and it may suffice if I merely refer to the probability that some rare instances of diverticula from the pharynx may be regarded as dilatations of portions of branchial fistulæ closed externally but remaining open within; and to the further probability that some congenital and infantile cysts, and even some ranulæ, may be derived from branchial canals closed at both ends and distended with fluid, as hydrocele in the inguinal canal may be formed by the filling of part of a *canalis vaginalis*.²

Of the branchial fistulæ in the external ears no account, I believe, has yet appeared in this country, and in foreign literature I have found descriptions of only six or seven cases, and some of those are very incomplete.³

The instances of which I have to tell are in the family of a member of our profession, an active well-formed man. He showed me a well-marked branchial fistula on the left side of his neck opening near the middle of the anterior border of the sterno-mastoid muscle, and often yielding a thin, clear, mucous fluid. His father, he told me, had an exactly similar fistula, and so had one of his sisters. Moreover, of his eight

¹ Heusinger, u. supra, and in B. xxxiii, p. 177.

² These matters may be studied in Heusinger. 'Virchow's Archiv,' B. xxxiii, p. 177; Roser, 'Eléments de Pathologie Chirurgicale,' traduit par Culmann, 1870, p. 177; Schede, as quoted by Schmitz, u. infra; and K. F. v. Heusinger, in the 'Deutsche Zeitschrift,' u. supra.

³ They are collected by Schmitz; 'Ueber Fistula Auris congenita: Inaug. Diss.,' Halle, 1873. The first and best case is by Heusinger, Virchow's 'Archiv,' B. xxix.

children four have, or had (for three of them are dead) similar fistulæ, secreting a "pure lymph," two of them having each one on the left side of the neck, and two of them each two on opposite sides symmetrically placed.

The prevalence of cervical branchial fistulæ in several members of a family, which has often been observed, is, in this instance, very marked; but that which is of more interest is that in this gentleman, and in his sister, and in five of his children, there are (or were) similar sinuses in the upper and anterior part of the helix of one or both ears.¹ Those which he showed me in himself, and which might be taken as examples, might easily be overlooked, for the orifice of each is minute, and the canal or fistula only just visible beneath the skin. The canal is little more than half an inch in length, and runs from behind forwards and a little downwards. It is less soft and flexible than the cervical fistulæ, and no secretion has ever been noticed; but he has observed that "on rubbing the part with pressure a peculiar waxy smell is communicated to the finger."

It might have been very hard to explain these fistulæ in the ears if their meaning had not been indicated by the co-existence of well-marked branchial fistulæ in the necks of the same persons or of members of the same family. Similar coincidence was observed in a case by Heusinger,² which I believe to have been the first recorded; and as the cervical fistulæ have been certainly proved to be the result of incomplete closure of the lower branchial fissures, so may these aural fistulæ be regarded as similarly due to incomplete closure of the upper or first post-oral fissure; or rather, of that part of it which is not utilised in the formation of the Eustachian tube, tympanum, and meatus.

So far as I know, these are the only cases in which the branchial fistulæ have been found opening on the helix.³ In

¹ A child lately born to him, the offspring of a second marriage, has neither cervical nor aural fistulæ.

² Loc. cit., p. 361.

³ Unless it be in a case, with malformed ears, related by Lincke, 'Handbuch der Ohrenheilkunde,' Leipzig, 1837, B. i, p. 615.

the other recorded instances the openings have been just in front of the lower and anterior part of the helix, or, in one doubtful case, in the lobule of the ear.¹

I believe that nothing is known of the causes of these malformations or of the conditions leading to the failure of the process by which, normally, about the end of the second month of embryo-life, the branchial fissures are completely closed. And as to the placing of the unclosed part on the helix, we can only guess that when the auricle, as part of a cutaneous operculum, grows up from the posterior and upper part of the second post-oral branchial arch, it carries with it a part of the cleft between the second and first arches, and this fails to close and lengthens into a canal. But I venture to believe that we shall gain a most exact understanding of these when more shall have been collected. They are, probably, not nearly so rare as they seem; and further illustration may be gained from the study of other malformations of the ear in which the branchial fissure is concerned. Such are, probably; the rare cases of complete transverse bi-partition of the auricle; and a case described by Feist, in which there was a linear depression in the helix, anthelix, and tragus, a trace of which appeared in a line on the cheek and nose, while behind the auricle were four small blind pits.

If we bring together all the cases of branchial fistulæ now known, it appears that there are instances of defective closure of every one of the post-oral branchial fissures.

The aural fistulæ, such as I have described, are defects in the closure of part of the first fissure; that is, of that part of the fissure between the mandibular and hyoid arches which is not used in the formation of the external meatus, tympanum, and Eustachian tube.

The upper cervical fistulæ are defects in the closure of the second fissure; that, namely, which is between the hyoid and the thyro-hyoid arches.

The lower cervical fistulæ may be regarded as similar

¹ All the cases, except that by Lincke, are collected by Schmitz, loc. cit.

² Quoted by Lincke, loc. cit.

defects between the thyro-hyoid and the fourth or sub-hyoid arches, or between the lowest of these arches and the upper boundary of the chest.

In association with these malformations it is very interesting to observe some of the instances of what are called supernumerary auricles. The natural auricle may be regarded as a developed and utilised opercular skin-fold of the first post-oral branchial cleft; and growths of skin, more or less resembling auricles, have been found, however rarely, in the situations at which are found cervical branchial fistulæ, the remnants of the lower branchial clefts.

The best case is that by Mr. Birkett,¹ of a girl seven years old, in whom there were two supernumerary auricles, as of ears, on the sides of the neck, one over the middle of each sterno-mastoid muscle, that is, in the ordinary position of the upper cervical branchial fistulæ. Mr. Birkett's account leaves no room for doubt that they were rudimental external ears; "the tissues of the lobes and of the fibro-cartilage of the auricle were clearly distinguished."

Mr. Holmes² also gives a case, showing association of a supernumerary ear and a branchial fistula. In a little child "a small pendulous body, something like the supernumerary ears, was attached to the skin near the hyoid bone;" and there was "a small sinus, about three quarters of an inch above the sternum, and leading down towards but not to that bone."

In one of Heusinger's cases,³ a flat, notched process of skin was attached just above the sterno-clavicular articulation, and at the inner edge of the sterno-mastoid muscle, and on raising it a well-marked branchial fistula appeared.

It thus seems probable, that in these malformations we may discern remnants of the serial homology, not only of the branchial fissures, but of their opercular skin-folds in the human subject. And similar facts are to be found in the less rare or even frequent occurrence of auricles, or

¹ 'Transactions of the Pathological Society,' vol. ix, p. 448, 1858.

² 'Surgical Treatment of Children's Diseases,' p. 140.

³ Loc. cit., p. 359; and for a probable though less marked case, p. 363.

portions of skin-like auricles, on the necks of sheep, goats, and pigs.¹

Dr. Allen Thomson² long ago pointed out a principle of great importance in the fact that malformations of the external ear are often associated with malformations of parts nearly connected with the first or mandibular branchial arch; for example, with hare-lip and cleft palate. And more recently, Virchow³ has illustrated the same principle, especially in the frequent coincidence of such malformations with supernumerary auricles or "auricular appendages." I have not found any instances of other malformations associated with the aural branchial fistulæ; but there has been a remarkable frequency of deafness in various degrees in the persons in whom these fistulæ have been seen. My friend, in whom and in whose family the cases I have related have occurred, describes himself as "dull of hearing" on the right side; his father, who had cervical fistulæ, was very deaf in one ear; his sister, who has an aural fistula, uses a speaking trumpet; his eldest daughter, who has two aural fistulæ, has

Chela
X

¹ Wilde, 'Aural Surgery,' 1858, p. 158; and especially K. F. v. Heusinger in the 'Deutsche Zeitschrift,' loc. cit. I believe that such supernumerary auricles are not found on other parts than those connected with branchial fissures. The skin-growth often seen near the true auricles, or on the cheeks, are probably growths of the same opercular skin-fold as the auricle from which they look like bits detached. Or they are auricles displaced, but still in the line or region of the mandibular arch, as in a case by Fielitz ('Stark's Archiv für Geburtshülfe,' B. ii, St. 1, p. 71), and in several of those collected by Virchow, 'Archiv,' B. xxx, xxxii. The case quoted by Wilde, and then by Virchow, and others from Cassebohm, of two supernumerary auricles on the back of the neck, was that of a double-headed Cyclopiæ monster, and these were the normal auricles appropriate to the two internal ears of the adjoining and partially fused sides of the heads (see Cassebohm, 'Tractatus sextus de Aure humana,' Halæ, 1734). And Wolff's case, often quoted from Voigtel ('Pathologische Anatomie,' 1804, p. 35), was that of a headless foetus in which an ear on the left shoulder was matched by a mouth on the right. Lincke, loc. cit., quotes from Sebenicius, an instance of ears on the upper part of the neck, but he does not say on which aspect of the neck, and I have not been able to refer to the original paper.

² 'Edinburgh Monthly Journal of Med. Science,' vol. vii, 1846, p. 420.

³ 'Archiv für Pathol. Anatomie,' B. xxx, and in a very remarkable case in B. xxxii, p. 518.

always been a little deaf, and more so in cold weather; and his youngest son, who has fistulæ in the neck but not in the ears, "has been decidedly deaf, but is now said to be improving." He adds, "I have a brother whose ears are most rudimentary. He is very deaf."

So in Heusinger's second case, the child with two aural fistulæ was brought to him for hardness of hearing, and was defective in discerning the direction of sounds. In Schmitz's first case, the father of the girl with aural fistulæ was completely deaf; her mother, sister, and aunt were very hard of hearing, and she herself had recently had otorrhœa and perforation of the left tympanum. In his second case the aural fistulæ were observed in a deaf old man.

It is hard to believe that so great frequency of defective hearing in the subjects of aural branchial fistulæ should be casual; and though, because the deafness was not congenital, we may hold that there was not, in these cases, any malformation of the interior structure of the ear, yet we may well believe that there was so much defect of texture as rendered some parts of the ear morbidly liable to disease. Allen Thomson and Virchow have shown how the disease of the embryo, whatever it may be, which hinders the development of the external ear, often causes other malformations by its extension over the mandibular branchial arch and the parts closely adjacent to it. Similarly, in these instances of diseases hindering the complete closure of the first or second branchial fissure, the morbid process seems to affect, in however slight a degree, the structures of the nearly related parts, and to render them, like all structures that have ever been diseased, specially liable to disease in later life.

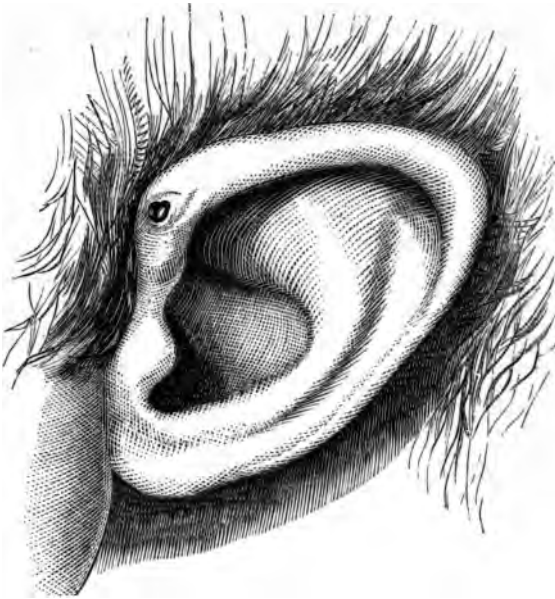
APPENDIX.

Since writing the foregoing paper I have received, through the kindness of Mr Cumberbatch, notes of a case which he has lately observed, and in which the traces of the branchial

fissure appeared on the helix, and by inheritance, but in the form of pits, not of canals.

Mr. C—, æt. 26, was deaf after scarlet fever in childhood. The right membrana tympani was thickened, dull white, and depressed anteriorly. The tip of the malleus-handle was somewhat enlarged. In the left ear similar appearances were more marked, and there were traces of an old perforation at the anterior lower segment of the membrana tympani. The tuning fork was heard distinctly when placed on the vertex of the head, and equally well on both sides.

The pinnæ of the ears were peculiar in shape, being somewhat triangular, and on the upper part of each helix, close to the skin of the temple, there was a small depression large enough to admit a No. 4 shot. (See woodcut.)



The patient's maternal grandmother, and her eldest daughter had similar pits in similar situations on their ears. But no cases of harelip, cleft palate, or other deformity, had

been known in the family, and deafness was not prevalent among them.¹

¹ Since the reading of the paper an essay on the subject has been published in the 'Edinburgh Medical Journal,' February, 1878, by Dr. Victor Urbantschitsch, "Concerning the so-called *Fistula Auris congenita*." In about 2000 aural cases he found twelve fistulæ. He refers to cases other than those which I have cited, and yet more references may be found in Jean Cusset's '*Étude sur l'Appareil Branchial des Vertébrés*,' 8vo, Paris, 1877. But the pathology of the branchial apparatus is overpassed when, in this essay, the cutaneous cysts found by the upper margin of the orbit and in the intermaxillary and other fissures are included among branchial cysts.

Through the kindness of Mr. Dalby and Mr. Mason I have seen two cases in which, with very imperfectly formed auricles and closed external meatus and small lower jaw, just as in one of Dr. Allen Thomson's cases, there was on each side a small shallow pit in front of the imperfect auricle. This pit or fistula might be likened to a branchial fistula, or else be regarded as an incomplete integumental portion of the imperfectly formed external meatus. Moreover, in Mr. Mason's case there was on each side, near what might be regarded as the upper edge of the incomplete helix, a smaller pit, about a line in depth, probably indicating a mere trace of a branchial fistula.

THE MICROSCOPIC ANATOMY OF THE SMOOTH TONGUE

("CHRONIC SUPERFICIAL GLOSSITIS").

BY

HENRY TRENTHAM BUTLIN, F.R.C.S.,
SURGICAL REGISTRAR TO ST. BARTHOLOMEW'S HOSPITAL; ASSISTANT
SURGEON TO THE WEST LONDON HOSPITAL.

(Received November 20th, 1877—Read January 8th, 1878.)

IN the following short paper I propose to give a description of the morbid anatomy of the smooth glossy tongue, called by Mr. Fairlie Clarke "chronic superficial glossitis." So far as I am aware, no description of the microscopical characters of this disease exists. I have examined three of these tongues which were removed because the disease in each case was complicated by epithelioma occurring after it had existed for some years. They were handed over for examination by Sir James Paget, Mr. Thomas Smith, and Mr. Langton, to whom I am indebted for permission to bring them before the Society.

Mr. Clarke's description of chronic superficial glossitis runs as follows:—"At the commencement of the disease some portions of the membrane present their natural appearance, while others are of a deep red colour and raw looking.

These patches are generally more or less oval or oblong. Their surface is smooth and glossy. They are either entirely denuded of epithelium or it is reduced to an extremely thin layer, and the papillæ are obliterated by distension. They are somewhat elevated and hard to the touch in consequence of interstitial thickening. . . . The tongue is large and swollen. . . . It is no uncommon thing to see superficial ulcerations on such tongues. . . . If this state of things goes on the mucous membrane of the whole tongue may become altered in character. . . . Supposing the more active mischief to be checked, the swelling subsides and the tongue resumes its normal size. . . . The patches that have been affected remain smooth and shining. The papillary structure has been impaired, and what is left is, in fact, cicatricial tissue."¹

This description accords, in the main, with the condition of the organs examined by myself.

CASE 1.—A draper, æt. 62, had had a bad tongue for many years. In June (1876) first noticed a small ulcer on the side of the tongue. Was operated on in October, when the tongue was large, generally indurated. Surface quite smooth, red, and glazed, especially at the anterior part. The surface of the tongue was also much fissured and cracked.

CASE 2.—A gentleman, æt. 57, who had suffered for many years from disease of the tongue, and in whom epithelioma had lately appeared. The general characters of this tongue were almost identical with those of the last. There was an epithelioma about two inches from the tip almost in the middle line.

CASE 3.—A man, æt. 40, who had suffered from syphilis, and was the subject of what is often described as a syphilitic tongue, namely, one of those perfectly smooth and glazed tongues without papillæ. There was also a large dirty-white patch on the surface of the tongue, not far from the tip. The epithelioma occupied the centre and left side of the organ.

¹ 'Diseases of the Tongue,' p. 159 - 161.

The microscopical characters were in each case essentially the same. The surface of the tongue was almost as smooth to the microscope as to the naked eye. The epidermis was generally diminished in thickness, being reduced to a thin, tolerably uniform layer. There was no trace of papillæ on the surface of the tongue. The epidermis consisted of two layers, a horny layer and a mucous layer; the intermediate layer of larger, more translucent cells, such as exists in the normal tongue, being generally absent. The cells in the horny layer were ill defined, owing to their flattened and horny condition. They had rather the appearance of the cells of the horny layer of the epidermis of the skin than of the tongue, a similarity still further borne out by the fact that they did not take up the colouring fluid, but remained yellow and shrivelled on the surface. The mucous layer was very distinctly, almost sharply, defined from the corneal layer. It was composed of round or oval cells, granular, nucleated, not varying *very* much in size or shape, except where the epidermis dipped down into the subjacent parts. The superficial layer of cells was flattened. The deepest layer of cells was generally arranged vertically to the surface, as in the normal condition. This portion of the epidermis was in many parts as uniform as the corneal layer, presenting no trace of papillæ even on its under surface; but in some parts it dipped down at irregular intervals into the subjacent tissues, so that a rough kind of papillary appearance, more like that of the skin than of the tongue, was produced. These downward processes were much more common and much larger as the epithelioma was approached. The cells composing them were much larger and more clearly defined than those of the mucous layer generally. The corium immediately below the epidermis was infiltrated with small round cells or nuclei to such a degree that little else was visible for a considerable depth than these cells and the blood-vessels. The blood-vessels appeared to be more numerous than in the normal condition, and larger than normal. The thickness of the corium was greatly increased, the muscular fibres in most of the sections being much far-

ther from the epidermis than in the normal tongue, and much more widely separated from each other. This thickening of the corium appeared to be due chiefly to the new formation of fibrous tissue, the characters of which were easily made out in the deeper layers. The muscular fibres appeared to be quite healthy, except those which suffered from the encroachment of the newly formed connective tissue. No other structures of the tongue were affected.

The disease thus appears to be limited to the mucous membrane of the tongue over a larger or smaller area. Expressed shortly, the changes consist in thinning of the epidermis with destruction of the papillæ and other appendages; in thickening and increased vascularity of the corium and infiltration of its superficial layers with nuclei or cells.

The first thing that struck me in the examination of these tongues was the great similarity in the conditions found with those described as existing in psoriasis and ichthyosis of the tongue. The most complete descriptions of the morbid anatomy of psoriasis linguæ are those of Debove¹ and Nedopil,² who say that the epithelial layer is a little thickened; that there is considerable thickening and sclerosis of the corium, with infiltration of small cells; and that the lingual papillæ are uniform, *mammelonné*, more like cutaneous papillæ, or are entirely absent. These descriptions are intended to apply equally to psoriasis and ichthyosis, between which no difference is made. Mr. Hulke³ speaks of having found gigantic papillæ in portions of a tongue which he removed and examined. Mr. Morris⁴ and Dr. Goodhart describe thickening of the epidermis and of the epithelial fibrous tissue, with crowding of "small rounded nuclei" in the latter. Mr. Fairlie Clarke⁵ speaks of "some increase of the thickness of the epithelial layer . . . some

¹ 'Le Psoriasis Buccal,' Dr. M. Debove (Paris, 1873).

² 'Arch. f. Klin. Chir.,' Bd. 20, s. 325 (1876).

³ 'Clin. Soc. Trans.,' vol. i, p. 1.

⁴ 'Brit. Med. Journ.,' 1874, vol. i, p. 231, 'Quart. Journ. of Micro. Science,' 1875, p. 96.

⁵ 'Med.-Chir. Trans.,' vol. lvii, p. 155.

enlargement of the papillæ, and in the submucous and muscular tissues there was a very abundant nuclear cell-growth."

Unfortunately the illustrations which accompany the above papers are not numerous; indeed, some of them are not illustrated at all, so that one cannot compare the sketches with the text in each case as one would like to do. In everything except the thickening of the epidermis the microscopic anatomy is precisely the same in psoriasis (ichthyosis) and chronic superficial glossitis. I have carefully examined the drawings attached to Clarke's paper and to Nedopil's. Comparing the thickness of the epidermis in Clarke's drawing with my own drawings of the epidermis of normal tongues magnified an equal number of times, the epidermis in Clarke's case appears not only not thicker but actually thinner than the normal epidermis. The most important of Nedopil's sketches is one which shows a portion of the diseased part of a tongue with the adjacent normal structures. An intermediate layer of cells is shown to exist between the surface or corneal cells and the rete mucosum, a layer similar to that of the normal tongue. But the very slight increase of thickening, if there be any, in the epidermis is due to the corneal layer.

I have only once had an opportunity of examining an ichthyotic tongue, and I then found *thickening* of the epidermis, with precisely the same conditions as those already described. There were not any enlarged papillæ, but where the white patches were present a thick layer of horny epithelium existed on the surface of the ordinary epidermis. I have, however, seen one case of ichthyosis in which the papillæ appeared to the naked eye to be considerably enlarged.

With regard to the nature of the disease whose anatomy has been described, it appears to be a chronic inflammation of the mucous membrane, which has gradually produced complete alteration in the characters of the epidermis and thickening of the corium and submucous tissue. The surface of the tongue is not reduced to the condition of mere

"cicatricial tissue," as described by Mr. Clarke, for some of the chief characteristics of such tissue are absent. There appears little or no tendency to contract; there is increased vascularity and thickening, not thinning of the corium. The presence of numerous cells and nuclei in the corium is merely an exaggeration of what is by no means uncommon in normal tongues. The disease is evidently closely allied to psoriasis and ichthyosis of the tongue; indeed, Nedopil speaks of the smooth glossy tongue as a condition preceding psoriasis, or as a lesser condition which gradually passes into complete psoriasis. I am not prepared to say what the exact relation may be, but as proofs of such a relation would point to the anatomy of the diseases, to the occurrence of epithelioma in both cases, and to the occasional appearance of a white patch upon the surface of these smooth glossy tongues.

DESCRIPTION OF PLATE IV.

Chronic Superficial Glossitis (H. T. Butlin).

FIG. 1.—Sketch with very low power (A 4 in.), to show the smoothness of the surface of the tongue and the diminution in thickness of the epidermis.

FIG. 2.—A similar sketch, from a point nearer to the epithelioma, showing downward prolongations of the epidermis.

FIG. 3.—More highly magnified sketch of a portion of one of the foregoing, showing the two layers of the epidermis, the infiltration of the corium and subjacent parts with cells. ($\times 40$.)

FIG. 4.—Another portion still more highly magnified, showing the thickened corium infiltrated with leucocytes. ($\times 60$.)

FIG. 5.—Much more highly magnified portion of the above, showing the characters of the various elements. ($\times 260$.)

Fig. 1.

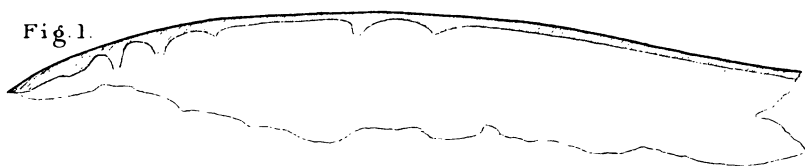


Fig. 2

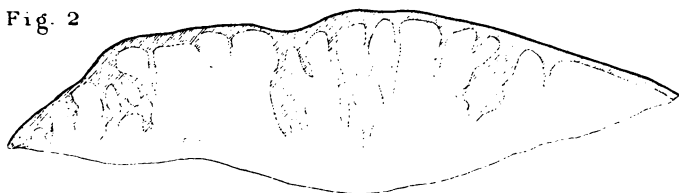


Fig. 5.

Fig. 3.

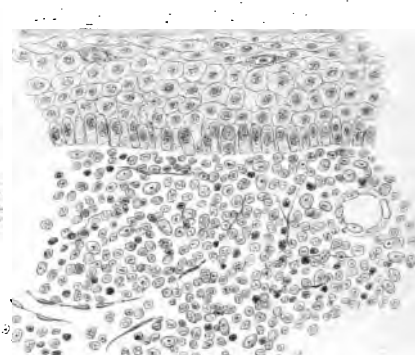
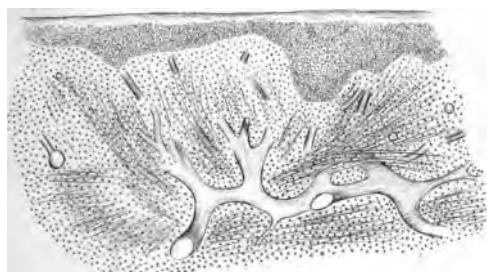
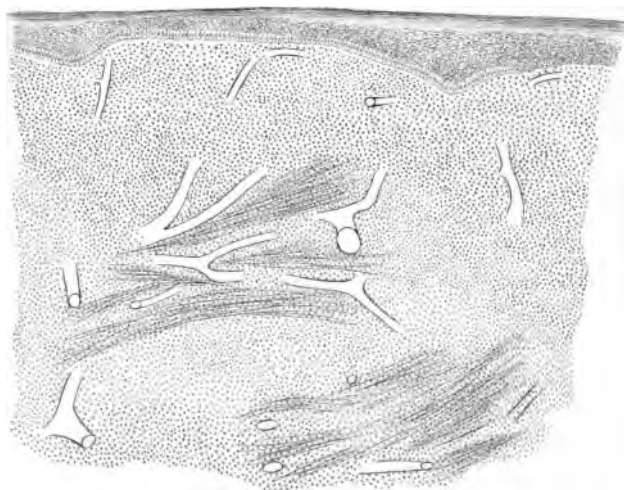
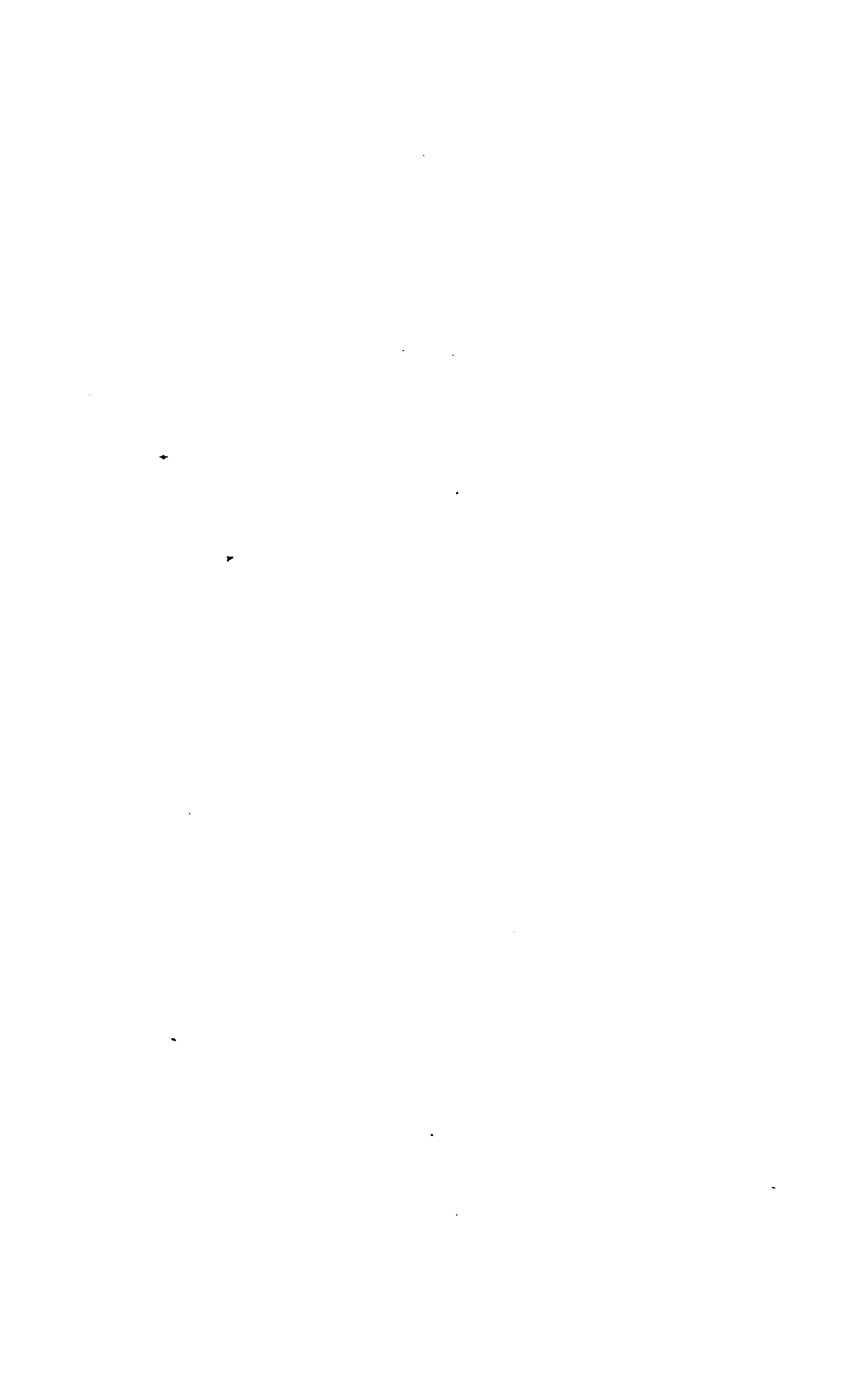


Fig. 4.





ON
MYXŒDEMA,
A TERM PROPOSED TO BE APPLIED TO AN ESSENTIAL CONDITION
IN THE
“CRETINOID” AFFECTION
OCCASIONALLY OBSERVED IN
MIDDLE-AGED WOMEN.

BY
WILLIAM M. ORD, M.D. LOND., F.R.C.P.,
PHYSICIAN TO ST. THOMAS'S HOSPITAL.

(Received October 16th—Read October 23rd, 1877.)

ON the 24th of October, 1873, Sir William Gull brought before the Clinical Society of London observations on five cases illustrating what he described as “a cretinoid state supervening in adult life in women.” The term cretinoid was avowedly used in that communication in a tentative or provisional way, and the cases were brought forward in the hope of directing attention to the state denoted by it, which Sir William Gull regarded as a substantive and definite one.

Having during the last twelve years had under care at least five cases of a similar kind, in one of which I had the opportunity of making a post-mortem examination, I propose to record two of them at some length, to make short reference to the others, and to draw inferences.

1. H. J—, æt. 54, came under my notice as an out-patient at St. Thomas's Hospital in 1871. She was a widow,

and had had two children, both then surviving. Her parents had not been subject to any nervous disorder or to any affection like her own. There was no reason to believe that they had suffered from any marked constitutional disorder. The catamenia were regular up to their cessation ten years ago. Her illness had commenced five years before. Up to that time she had been active, and not stouter than the average.

She had, when first taken ill, been very anxious in connection with the fatal illness of her husband. The first signs of illness were fits of shivering during her work. These were followed by the passage of bloody urine, as she believes, on several occasions. Then her hand became "dead," to use her own expression, when she used her needle; a great addition to her trouble, as she had to work very hard to support herself and her helpless husband. Later on she became "weak-headed," would be stupefied by a glass of beer at luncheon, experienced a general loss of muscular power, and was always falling asleep. After that she had constant pain and weakness in the back, so that she began to stoop considerably; her speech became slow and difficult, and a gradual swelling of the skin of the whole body set in; the skin of the face, and particularly of the eyelids, becoming thick, semi-transparent, and waxy. When first seen she exhibited in a very marked form the character of skin described by Sir William Gull. The face was generally pale, but had the delicate blush on the cheeks. The eyelids, the lower in particular, were swollen and ridged, so as almost to hide the eyeball, and at the same time to hang down flaccidly on the cheek; they were remarkably translucent, and had somewhat the look of the eyelids in acute Bright's disease. But they were wrinkled on the surface instead of being smooth, and did not pit on being squeezed between the thumb and forefinger. The nose was swollen, broadened and flattened, the nostrils being widely expanded; the lips were swollen, expressionless, and pressed together, the mouth being a simple horizontal slit, opening but slightly in the vertical, more freely than usual in the transverse

direction. The skin was again swollen into folds on the sides of the face and below the chin. The hands were swollen, the fingers looking very clumsy, and much limited in range of flexion and extension; very fairly describable as "spade-like."

The skin over the whole body was singularly dry. On the limbs and trunk it was harsh and rough to the touch; the hairs were feebly developed, and no trace of fatty secretion could be found. The skin was everywhere sensitive, but the limit of confusion in tactile discrimination was everywhere wider than the average. The same sort of swelling observed in the skin was visible in the fauces.

Her mode of speaking was very characteristic. The voice was not harsh or hissing, but dull, leaden, and without intonation, reminding one to some extent of the voice in an early stage of quinsy. When about to speak she closed her lips throwing the under one forward, made a movement of swallowing, and then with much widened mouth commenced inhalation, at the same time expelling air explosively through the nose. The swallowing appeared to be necessary to move the soft palate. The speech was slow and measured. It was noticeable that whatever she did was done slowly and after delay. With the exception of some dimness of sight and the slight deficiency of tactile sensation her senses seemed perfect. But there was a marked slowness of perception, and a marked slowness of response of muscles to voluntary or reflex nerve-impulse. She stated that she could not act or think quickly, that her thoughts would only come slowly; that where any operation, such as dressing, took her half an hour formerly, it now took her two hours, so that she was perpetually behindhand with her work. Although her temper was placid, this was a constant source of distress to her. She could walk only very slowly, and the knees often gave way suddenly. She was consequently very nervous when out of doors, and was always expecting that people would run against her, she being unable to get out of the way; in fact, she was subject to a sort of waking nightmare. She very often had headache, and felt always

tired, so that her life was utterly wretched. For some time she felt a strange taste in her mouth, which seemed to affect all food; it was partly sour, partly like the taste of blood. She often noticed a foul smell (as of bad breath) in her nostrils. The appetite was bad. The tongue was large and clean, not marked by the teeth, although too large for the mouth. The heart was feeble in its action, and not hypertrophied; there were no murmurs, and no important change of accents in the sounds. The breath was short on exertion or excitement, but not at other times, and there was no orthopnoea. The liver and spleen were not decidedly enlarged, but were somewhat tender, and undoubtedly firmer to the touch than normal. There was apparently a good deal of subcutaneous fat in all parts of the body, particularly in the neck, above the clavicles; and the thyroid body could not be felt. The blood examined under the microscope appeared healthy. The urine was examined repeatedly. The specific gravity was rather low, ranging between 1008 and 1017. The quantity was not materially increased. It contained no trace of albumen in the years 1871 and 1872.

In 1875 all the above conditions existed, in conjunction with increase of debility and languor.

At the beginning of 1876, on her appearance after a long absence, the urine was found to be albuminous, and there was true œdema of the hands and legs. In January, 1877, her state had undergone much change.

The complexion was pale yellow, and in the flush on the cheek were many enlarged vessels. With the occurrence of ordinary anasarca there was some abatement of the old nervous symptoms. She put out her tongue, spoke, and moved more quickly. The dryness of the skin was as marked as ever; but under any excitement she broke into excessive though short-lived perspirations. The skin of the body and limbs was shining, and exhibited flat smooth spaces, alternating with wrinkles and furrows. She "had lost that dreadful sinking in her inside," but was easily put out of breath, and could not lie down with comfort. The heart was decidedly enlarged; the first sound at the impulse was

reduplicated, and so was the second sound at the base, between aorta and pulmonary artery. The second sound was greatly accentuated over the aorta. The arteries were everywhere hard and tense, and the right radial was dilated and irregular in form, as though from atheroma. Tracings were taken of the left radial, obtained with difficulty on account of the toughness of the skin, and obscured by irregular respiratory and muscular movements.

Hawksley's surface-thermometers applied to the surface of the body gave 94.9° for the temperature below the right mamma, and 95° below the left; the temperature of the ward 60° ; of the axilla 97.2° , and of the mouth 98.8° .

She complained of feeling the cold much, of bad sleep, broken by frightful dreams, but less of the bad taste and smell.

The urine was 1004 sp. gr.; was faintly acid, pale, clear, and contained a little albumen; quantity increased. The symptoms attending destruction of the cortex of the kidneys were clearly developed, and had taken the place of many of the older and, as I believe, quite distinct symptoms.

And these late symptoms increasing in severity, ended fatally by breathlessness and exhaustion at the beginning of March, 1877.

Her axillary temperature continued low throughout January, and up to her death, falling many times to 95° , and not exceeding 97.6° till four days before her death, when it rose to 99° .

The full notes of the post-mortem examination are appended to this paper, and I will here mention only one or two important details.

1. There was œdema of the skin generally; but the cut surfaces yielded less fluid than their appearance would promise.
2. There was much serous effusion in the pleuræ, pericardium, and peritoneum.
3. The heart was of large size, weighing sixteen and a half ounces; the left ventricle hypertrophied, the wall being an inch thick; valves practically healthy.
4. The arteries were everywhere thickened, the larger ones atheromatous.

5. The cortical substance of both kidneys was much wasted and granular both on surface and on section. The renal arteries were thickened and atheromatous; and there was thickening of their outer coats.

6. There was a firm, almost solid œdema in many parts, *e.g.* in heart, soft palate, larynx, stomach, and neck of bladder.

7. The brain showed very considerable degeneration of the larger arteries.

In preparing portions of the organs for microscopic observation it was noticed that the skin in particular retained its œdematous condition even when cut up into small fragments, whereas the skin of dropsical patients collapses when so treated, owing to the ready draining out of the serous fluid. The skin of the feet was removed and examined by my colleague Dr. Cranstoun Charles and Mr. Shaw, with reference to the quantity of mucin to be detected, the first microscopic views giving rise to the idea that the normal mucin-yielding inter-fibrillar cement was here in excess. These portions of skin being subjected to parallel processes yielded equal and considerable quantities of mucin. Equal portions of skin from the foot obtained from non-œdematous bodies, treated in exactly the same way, yielded less than a fiftieth of the quantity obtained from the foot-skin of H. J.*

The description of the processes used by Dr. Charles and of the microscopic appearances found in various parts are set forth at the end of the paper, together with the full post-mortem report.

For the drawings of microscopic appearances I am indebted to the skill and kindness of my colleague Mr. Charles Stewart, of whose accomplishment in histology it is unnecessary to speak. They were made with the aid of the camera lucida, and the nuclei are individually of the exact size and distribution of the nuclei in the stained sections from which the drawings were made.

* Since the paper was read, the skin of œdematous bodies has been subjected to the same processes, with the result that no increase of mucin was found.

The second case which I shall quote is that of a lady, æt. 36, who came under my notice in 1863, and remained more or less under my care till her death in 1870.

When I first saw her she was extremely stout, puffy in the skin, and unwieldy. Her face had a tranquil expression, but the surface was waxy and transparent, the eyelids were greatly swollen, and she had the delicate blush on the cheeks. At first sight she appeared to be suffering from renal disease, but on examination no evidence whatever of its existence could be found. The swollen transparent state of skin prevailed in all parts of the body; the lips were blubbery, the tongue was swollen and was protruded slowly, the fauces were swollen without congestion, and the neck was creased with gelatinous-looking folds. The hands and feet were bulky and broad in proportion to their length. The vulva and os uteri displayed the same condition to a marked degree, and the rectum and anus owing to the soft swelling of the mucous membrane offered a serious obstacle to defæcation. It may be remarked that the condition of the genital organs did not prevent conception. She was twice pregnant within the seven years, and on each occasion went her full time. Although her weakness caused great anxiety, her labours were not unfavorable, excepting that that there was some excess of hæmorrhage. The skin except on the face was rough and indisposed to perspiration. These conditions which had been slowly developed during the three years preceding my first knowledge of her, were attributed by her to a very rapid succession of pregnancies. Before marriage she had been slight and active. Her parents were both living, and there was no unhealthiness in her collateral relatives. As in the former case there was no history and no indication of syphilis.

Associated with the foregoing were three marked sets of symptoms:—1st. A remarkable slowness of thought and action; 2nd. A distinctively slow articulation; 3rd. A slowness in muscular action, and an incapacity for the maintenance of muscular effort.

1. Under the first head it must be noted that, though

placid in her manner, and absolutely amiable in her temper, she was acutely conscious of her slowness of thought, and painfully prolix in dilating on it. As the head of the household she endeavoured during her husband's necessary daily absence to fulfil her duties, but although constantly employed scarcely accomplished anything. The mere act of dressing cost her between two and three hours. Her memory was good, her ideas perfectly clear, but she seemed like a bulky mass slow to respond to external forces, and to take on movement. And her momentum when once set in motion was equally noticeable. The current of her thoughts having been set to flow in any one direction went on flowing, and the digressions thus produced were among the causes of her administrative inefficiency. A question edged in at a pause for breath was, so to speak, swept away by the current, but was not lost, being answered when the whole of the thoughts which she wished to express were displayed, sometimes after an interval of two minutes. She wrote well, calligraphically; her letters were long and diffuse, but always accurate.

2. In speaking, she commenced by closing her lips and swallowing; her voice was nasal, with defective intonation; and while articulating, she often made a partial act of swallowing, and ejected air by the nose. There was a perfect resemblance between her way of speaking and that of H. J.

3. Her walking was a very painful process. She was obliged to move very slowly, and at each step a quiver ran through her whole frame as though the muscles were behind hand of her will, and were not in perfect concert with one another. There was no tremor however, and nothing at all like locomotor ataxy. The movements were like the quivering movements of a person pacing slowly while deeply engaged in thought, and the ultimate principle involved, appeared to me to be the same. As it would be under mental abstraction, there was a want of free response on the part of the central nervous system to influence from without. This want of correspondence sometimes went so far as to cause heavy falls, a step being taken by the agency of one set of muscles before

another set were in readiness to carry on the chain of movements. She sustained in this way a fracture of both ligamenta patellæ, one at one time, one at another, the extensors of the thigh waking up just in time to do the mischief.

As time grew on, the expression of her face became very distressing. It was certainly cretinoid, the swollen lower lip protruding and depending, and at times allowing of the escape of saliva. Her head hung down with the chin on the chest, owing to the inability of the muscles of the neck to maintain their action. When the head was raised after several oscillations, by the slow action of the proper muscles, or by the aid of the hands, it often fell helplessly backwards or to one side in such a way as to make bystanders shudder lest the vertebral ligaments should share the fate of the ligamenta patellæ.

There was no affection of the senses or of the viscera, so far as could be made out, till about six months before her death, when the urine, hitherto perfectly normal, began to be albuminous, and the vascular symptoms of renal disease were rapidly developed. She died comatose, and no examination of the body was obtained.

In three other cases I have witnessed a similar combination of symptoms. The patients were all women between forty and sixty years of age. In two of them the urine became at length albuminous, but not till the anasarca appearance had existed six years in one, and nearly ten years in the other case. All three of these have passed out of my sphere of observation. But in all, the gelatinous appearance, the harshness and the dryness of the skin were well marked; in all there was the slowness of thought and action and the slow, painful enunciation.

The cases agree, in most respects, very closely with those narrated by Sir William Gull; in fact, I am inclined to think that two of them may have come under his observation. It is evident that Sir William Gull has used the word cretinoid, in relation to his cases by way of labelling them, and of putting them aside thus labelled for further comparison and more minute investigation.

The opportunity which I have enjoyed of recording a post-mortem examination of a case of the kind enables me to carry, at least, a suggestion as to the pathology of such cases a step further. The suggestion is to the effect that a jelly-like state of the fibrillar or white element of connective tissue is the essential and common condition of these several cases, and that nervous disorder and vascular change are definitely consequent thereon.

The apparent œdema so well marked in the face, and not more marked, indeed, at first less evident, in more dependent parts—an œdema which is undisturbed by pressure or change of attitude, which is exempt from the laws ruling the distribution of fluids in closed vessels—appears to me an indication of the presence in the skin of either a jelly-like interstitial material or of tissue in a jelly-like state. The microscope (see Plate V, figs. 1 and 2) shows that the interstitial spaces of the cutaneous tissues are large, and that the fibrillar element of the connective tissue is everywhere swollen, while the nuclear element is highly developed, and the nuclei larger than the average. The swelling of the fibrillar element is associated with unusual definition, separability, and size of the fibril. This is well seen in the corium generally, but is brought out most clearly in the investments of glands and of hair-sacs, and in the coats of vessels (Plate V, fig. 1).

In the coats of arteries, the adventitia can often be seen swollen to three or four times its proper proportion, with the fibrils unusually well defined, as though separated from each other by a swollen interstitial substance. And the middle coat, also much thickened, has a much greater increase of matter not stained by logwood than of nuclei. There are, perhaps, more transverse nuclei than should belong to an artery of a given calibre, but they are imbedded at much wider intervals than is natural in the unstained surrounding matter. In many sections these two overgrowths appear to have led to the obliteration of arteries, for there are many round *areæ* which look like arterial structures without a central cavity—*areæ* which, in size, would corre-

spond to capillary arteries. In the kidneys a similar thickening, growing inwards from the capsules of Malpighian bodies, can be seen encroaching in various degrees up to obliteration upon the contained glomeruli of capillaries (Plate VI, fig. 1).

4. In the liver an enormously disproportionate swollen connective tissue separates the cells from one another, and evidently encroaches on them, and tends to produce atrophy in them (Plate VI, fig. 2).

In the thyroid gland the alveoli are compressed, and mostly annihilated, by a growth of the kind which can here be seen in great perfection around the vessels; and in the muscular tissues, particularly in the heart, the same sort of excess of cement, with induced attenuation of the muscular elements, is clearly to be recognised. A natural condition very closely resembling this is to be seen, as Dr. Harley has reminded me, in the umbilical cord, where the connective tissue has also often a dropsical look, from excessive infiltration with the mucus-yielding material. In conditions of this kind arises, as I infer, the inaction of the skin observed in all the cases. This may be explained in two ways, 1st, by the manifest atrophy of the sweat glands; 2nd, by alterations in the nerve-endings in the skin. Where these nerve-endings can be observed, it is seen that they are enveloped in soft transparent substance, so as to be padded and removed from the ready operation of incident impulses, tactile, thermal, or chemical. They are so placed, therefore, as to receive fewer impulses, and to receive such as reach them more slowly, so that the first stage of a bradæsthesia is set up. Observation in the case of H. J— showed that there was no material loss of accuracy in perception, while there was a remarkable slowness of response to external and volitional impulses. It is possible that a parallel state of things prevailed in the nerves and nervous centres, but, though I have submitted sections of the latter to the microscope, I cannot say positively that they warrant such a conclusion. The existence, however, of a condition of the skin interfering with the natural exposure of nerve-ends to stimuli appears

to me in itself an explanation of most, if not all, the characteristic nervous phenomena above related. One most essential condition of the health of the central nervous system is that it receive its due amount of stimulation through the skin, and receive it regularly. The stimulation is probably complex. Light appears to be one stimulus, variation of temperature another, variation of dryness and moisture another, the chemical quality of air or water another.

Experience demonstrates the evil result of a restricted indoor life, the benefits of out-door exercise, of bracing air, of the exposure in journeyings by sea. Proceeding upon this basis I would argue that, supposing the cutaneous nerve-ends to be sheathed unnaturally with a coat of jelly, there would result a failure of the natural stimulation of the centres—a failure sufficient to produce lethargy and inertia of the great centres, with slow mobility and slow response as results.

If the skin be alone regarded, the morbid state called sometimes sclerema, sometimes scleroderma, may be held to be allied to the state here described. The microscopic appearances are certainly analogous; but in sclerema the affection is limited, not general; is varying and not progressive; leaves textural changes not observed in myxœdema; and the swelling is hard instead of pulpy. At the same time I may observe that in the most typical case of sclerema adultorum which it has been my fortune to see, there have been nervous symptoms of a decided kind. When the skin was tense, hard, and dry, there was torpor, slow thought, slow action, and a perpetually oppressive sense of inadequacy of power and energy to the needs of daily life. These symptoms passed away when the action of the skin was established, and in a long course of observation were found to vary with the freedom of perspiration.

Again, the result of varnishing the skin of animals, as practised by Becquerel and Breschet, Valentin, Edenhuizen, and others, may be noticed for sake of comparison. When the skin is covered with a gluey insulating material the temperature falls rapidly and death follows. Even if only

one sixth of the skin of the whole body is thus insulated the fatal result is produced. This, excluding ideas of poisoning by retained secretion, is due either to rapid loss of animal heat through the varnished surface, or to loss of the tonic influence maintained by exposed periphery; to the latter mainly, as from analogy, I believe; and as the failure of the introduction of heat from without to do more than defer death helps to prove.

The want of free cutaneous secretion is to my mind, as much a result of the insulation of nerve ends as of the compression of the glands. A free cutaneous transpiration is generally held to be a condition and sign of health. The "sweating palm" is called by Shakspeare "the precedent of pith and livelihood." And certainly this phenomenon is part of a chain having for its chief link a vigorous health of the central nervous system. A day of fatigue, a night of excess, dry up the sweat and roughen the skin, for all that the glands still exist, and independently of all external agents affecting the surface of the body. Then, moreover, it comes to pass, that the induced condition of the skin is reverberated upon the centres, in feelings of distressing tension, and fulness, and heat, lasting till the internal disorder having passed away, the skin resumes its proper functions, and with the sign of local health the general ease returns.

To proceed, holding in view suggestions of cretinism; the hands, in all the cases noticed, have displayed the very converse of the conditions belonging to local and general health. Sir William Gull has drawn special attention to the swelling, broadening, and shapelessness of these members. He calls them "spade-like," an epithet which is well justified by their shape and by their want of natural movement and expression. I trust that I shall not be taken to task too severely for claiming *expression* as a property of hands. At all events, I have the support of the physiognomist and of the artist in believing that the hands are in their way an index of the character and reflex of the habits of the man. The movements accompanying passion, the ger, the clutch of avarice, the hearty grasp

of friendship, the soft pressure of entreaty, mould the lines of this feature in proportion as they recur often, or as ideas related with them often arise in the mind; the service to which the hand is put, again declares itself not only in the acquired texture of the surface, but also in the development of special qualities of strength, mobility, sensitiveness, delicacy, &c.,—in the attitudes, which in rest, remain as shadows left by activity.

The hands of the Crétin, as described in the 'Report of the Sardinian Commission of 1848,' not only have the spade-like shape, but are also covered "*d'un cuir rude et épais.*" The report goes on to say, that "*Il paraît que pour lui (the crétin) la main est un instrument uniquement destiné à saisir, et non à d'autre chose.*" A few crétins acquire by long practice a marvellous dexterity in twirling sticks between their fingers, but none arrive at the power of distinguishing by touch a silk from a cotton stuff, or the like. The hands in crétins are, again, but the type of a general indifference of the whole surface to external impressions. Crétins are mostly indifferent to great cold and great heat; they sweat rarely, and do not care to adapt their clothing to variations of climate. In the case of H. J—, a certain indifference of the skin to atmospheric conditions was well seen. It is true that she was always much depressed in winter, and was conscious that cold weather increased all her troubles. It is also however true, that in cold weather, with windows open near her, she commonly lay with the shoulders and upper half of the thorax covered only by a chemise, which was more often open in front than not. There was no sensation of cold which induced her to cover herself well with the bedclothes. It has been pointed out that perspiration was rare in her, and in the other patients here noted, and, what I do not find in the Sardinian Report, a general dryness, harshness, and coarseness of skin prevailed, more notably in the limbs than on trunk or face. The muscular movements again have in the cases described a decidedly crétinoid character. The attitudes and movements described in the second case were chronicled by me before it occurred to

tinism. The Sardinian étién as follows:—"Ils s'ivrognes, tiennent le *e penchée*, traînent les abers à la rencontre du dly an exaggeration of was not approached in ness of movement and a one developed.

mediately in question, my tion of symptoms are ling of the connective ng in an overgrowth of the fibrils of the white ngly I propose to give ion.

rtant mode of operation he symptoms associated ripheral termination of nuscular nerves, with a ee reception of impres- l in the external meatus e auditory nerve. That usual, and the central altered state of the skin, a, a state of intellectual

lethargy and a slowness in co-ordination of movements are necessary consequences. In this chain, slow use, partial disuse, and numbness of faculties are links of one kind, and the constant retardation of guiding sensory impulses, a link of another, so that supposing the myxœdema to be constant, the nervous degradation tends to be progressive. The train of symptoms leading to the fatal termination commences in the encroachments of the myxosis upon vessels and upon the secreting elements of glandular structures. Both fatal cases showed at the last, the symptoms associated with renal disease. And in the case of H. J—, the encroachment of the myxosis upon Malpighian bodies in kidney, and upon hepatic cells

has evidently proceeded to an extent involving very large interference with the functions of the organs in question. The same process can be seen at work in the practical destruction of the thyroid gland, in encroachment in the glandular tissues of the skin, and in a sort of gelatinous embedding of the vessels, more particularly of the arteries.

Many circumstances in the second case indicate that there was weakening of the toughness of tendons. Both ligamenta patellæ were broken in succession, and the ligaments about the head and neck were evidently lax and yielding. The alteration in the elements of connective tissue would explain this.

If this condition be admitted as an explanation in the cases before us, the parallelism between many of the symptoms associated and certain symptoms of cretinism, the parallelism which has led Sir William Gull to call these cases cretinoid, may justify me in suggesting that it may be possible to trace some similar cause in operation as the beginning of cretinism itself.

1. Cretinism is, according to all authorities, a progressive disease. At the time of birth there is, according to many authors, not much difference between a crétin and a non-crétin; so much so that it is not commonly a point which can be decided until the end of five or six months. That is to say, the unequal and irregular development of the head, the coarseness of the features, and the deformities of the body, are far less marked at birth than afterwards. However much this statement may bear qualification, it is abundantly shown that the perfect development of cretinism does not take place till the age of from four to seven years, according to different observers, while at the same time no healthy child becomes a crétin after such an age is passed.

2. Foderé, as quoted by the Sardinian Commission, asserts that those infants which, without presenting a congenital goître, are altogether destined to become crétins, have a body extraordinarily voluminous, and a head and hands of disproportionate size, and are mostly *œdematous*.

3. A "subcutaneous œdema," to use the expression of the

Sardinian Commission, is of frequent occurrence in adult crétins.

4. While goître is common in the districts where cretinism is common—their association in one individual is not invariable. Many goïtrous persons are not crétins; about one crétin in three is goïtrous. In the cases under observation there was notable diminution in the size of the thyroid gland, and the practical annihilation of the gland in H. J— has been described. In respect of this we may be reminded of Mr. Curling's¹ cases of absence or atrophy of the thyroid in idiots in association with great deposits of fat in the supra-clavicular region on both sides. The relation between goître and cretinism is ably and fully discussed by Dr. Hilton Fagge in a paper in volume liv of the 'Transactions' of this Society, "On Sporadic Cretinism occurring in England." I may remind the Society that this paper brought out more fully the principle which seemed likely to follow from Mr. Curling's observations, namely, that while goître was more or less associated with endemic cretinism, the thyroid gland was actually absent or atrophied in sporadic cretinism occurring in this country; and that Dr. Fagge infers the existence of a direct antagonism between goître and cretinism. It is unnecessary to reproduce his arguments, but it is obvious that the case of H. J— is one more in favour of them.

If Dr. Fagge's hypothesis that the presence of goître is actually a safeguard against cretinism, and that the atrophy of the thyroid is a possible cause of the sporadic cretinism of this country, be true, it may still follow that there is an ultimate cause present in the one case, and not in the other, upon which the growth or degeneration of the thyroid may depend.

In the cases now under consideration nothing is known of the state of the thyroid in early life; the occurrence of the swelling in the skin had certainly not come on till long after adolescence—till long after the intellect and the brain have undergone their complete development. The nervous symptoms here are accordingly such as would be induced by

¹ 'Med. Chir. Trans.,' vol. xxxiii, p. 303.

partial numbing of the senses, by the sleep or torpor of a central nervous system already built up and capable, in proper conditions, of exerting full activity. Supposing, however, the œdema to begin with life, the position of the central nervous system is different. It should, in natural conditions, be built up, be evolved in great part by the agency of impressions from without. As the deaf child is mute, so the child with insensible skin loses the education upon which the establishment of relations with the world of intelligence depends. The uninformed brain, the sensorial system without exercise, remain shapeless. External influences should, as was fabled of the bear with its cubs, lick the shapeless central organ into shape; and when they are hindered from contact, the brain lies like a polype in the water. Life is possible, because the machinery of organic sensation is not entirely wanting, but an organic life and no more; so that year by year during childhood the crétin, on this supposition, would, as the crétin, in fact, does, depart further and further from the type of healthy humanity.

Lastly, in giving a name to the condition observed, I trust that I may be considered to have shown reason for the new coinage; but the name is only intended to represent the condition, and does not profess to involve an explanation of its causes. Whether the mucous œdema be a degeneration, an arrest of development, or an introduction of new material, is not, at present, a question ripe for discussion; and though I should be grateful for suggestions on these points, I do not propose at present to express any opinion of my own.

Report on Mucin from the Skin by Dr. CRANSTOUN CHARLES.

Specimen of Mucin, No. 1. Mode of preparation.—The skin of both feet of Mrs. J— was cut into pieces, and divided into three nearly equal portions, α , β , and γ .

α was digested with water for several days; the filtrate from this was treated with an excess of acetic acid, let stand some hours, the precipitate separated on a filter and washed

first with water acidified with acetic acid, and then with pure water. This washed precipitate was next left for twenty-four hours in lime water, the solution filtered, and the precipitate again thrown down by excess of acetic acid. To purify the precipitate thus obtained it was washed successively with acidified water, pure water, alcohol, and ether, and then dried over a water bath.

The process is that employed by Eichwald for the separation of mucin from *Helix pomatia* and from tendons. ('Ann. Chem. Pharm.,' Bd. 134, s. 177).

β was left in methylated spirit for three days, in lime water for two days, then filtered, and to the filtrate acetic acid added in excess. The precipitate was separated and purified as in α .

γ was digested at once with dilute baryta water, the dissolved mucin precipitated by acetic acid, and purified as before.

The body obtained by the above three methods was sensibly the same in each case in properties and in appearance, and nearly equal in amount; it corresponded in its reactions to the mucin of Scherer, Eichwald, and Staedeler. (See Hoppe-Seyler, "Handbuch der Phys. Chem. Anal.," 'Ann. Chem. Pharm.,' Bd. iii, s. 14, Gorup-Besanez, ('Handbuch der Phys. Chem. Anal.')

By operating in like manner Hilger obtained similar mucin in small quantities from the skin of *Holothuria*. ('Arch. f. d. ges. Physiol.,' iv, s. 336.)

Specimen of mucin, No. 2 (from healthy subject).—This was prepared by Mr. Shaw, under my directions, from the skin of the feet of a healthy adult body. The methods employed were the same as the above.—T. CRANSTOUN CHARLES, M.D.

Report of post-mortem examination.

By Dr. W. S. GREENFIELD.

Hannah J—, æt. 58, widow, admitted into Alice Ward,

January 2nd, 1877, under Dr. Ord. Died March 7th, 1.45 a.m. Examined March 7th, 2 p.m.

Body of female of short stature. Rigor mortis well marked. Great toe joints healthy. Considerable anasarca of face and lower limbs, which scarcely pits on pressure; but on cutting into the skin the fat is found to be considerably œdematous; and watery fluid can be squeezed out of it.

Pleuræ.—Right contains a few ounces of fluid; left about the same; (quantity not measured).

Pericardium somewhat widely exposed and contains nearly a pint of clear serous fluid. Left lung considerably compressed by the distended pericardium.

Heart.—Weighs $16\frac{1}{2}$ oz.; of large size. Some general opacity of visceral pericardium, especially about great vessels at base. A patch of milky thickening ("plaque laiteuse") at base of right ventricle; two or three at base of left.

Left ventricle mainly enlarged, globular; apex rounded; cavity somewhat dilated. Wall (in semi-contracted state) measures at centre 1 inch; near base $\frac{3}{4}$ inch; near apex $\frac{5}{8}$ inch. Muscular tissue rather pale, tough.

Left auricle slightly dilated, otherwise normal. Left auriculo-ventricular orifice $3\frac{1}{2}$ inches in circumference; mitral valve competent, and except slight atheroma of anterior segment (in usual position near base) normal; no thickening of edges.

Aortic orifice only $2\frac{1}{8}$ inches in circumference (normal $2\frac{5}{8}$ to 3). Aortic valves competent and natural, free from notable thickening.

Aorta.—Arch of aorta somewhat atheromatous, not obviously dilated.

Great vessels.—Left vertebral artery given off separately from arch, between left carotid and subclavian. Great vessels extremely atheromatous, especially the left carotid, the patches of atheroma greatly thickened, prominent, and superficially of somewhat gelatinous appearance.

Right side of heart fairly healthy. Right auriculo-ventricular orifice $4\frac{1}{2}$ inches; pulmonic $3\frac{1}{2}$. Valves normal.

Branches of pulmonary artery here and there very slightly atheromatous.

Lungs.—Right 13½ oz.; left 9 oz. Both lungs, but especially left very emphysematous; upper lobe of left lung slightly œdematous; lower compressed, free from œdema. Right free from œdema.

Larynx.—Mucous membrane folds at entrance in, and also vocal cords, œdematous.

Liver 3lbs. 5 oz.

Gall bladder contains a large quantity of inspissated bile; and some normal bile.

Capsule here and there slightly thickened; with some small points of fibroid thickening, in places invading the surface of the organ, with some resemblance to grey granulations.

Hepatic tissue apparently normal.

Spleen 3 oz., small, firm, tough, congested.

Stomach.—Mucous membrane somewhat thick and opaque. Along both greater and lesser curvatures, and especially near pylorus, are numerous, small, slightly depressed, smooth cicatrices, the edges of which are vascular or slightly pigmented. (Small mucous ulcers.)

Kidneys, 9½ oz., of fairly normal size; capsule somewhat thick and opaque, but separated pretty easily from cortex, the thickening somewhat irregular, but not obviously laminated.

Surface of kidneys generally fairly even, though minutely granular; but scattered over the surface are numerous small well-defined depressions, smooth at base. These on section are found only to involve the superficial part; they do not extend through the whole thickness as scars.

Substance of organs somewhat firm, slightly tough, and both surface and section granular. Cortex generally but uniformly wasted, and its arrangement not much distorted; it contains a few small cysts.

Minute arteries in kidneys generally considerably thickened.

Renal arteries much thickened, very atheromatous; the outer coat also apparently thickened.

Bladder.—Some slight signs of chronic congestion about outlet, mucous membrane swollen, congested, and slightly pigmented.

Submucous tissue generally œdematous.

Brain.—Arteries at base extremely degenerated and atheromatous; so also all the visible arteries on surface, even to most minute, present extensive atheroma. The vessels at base appear also to present calcification of middle coat.
—W. S. G.

DESCRIPTION OF PLATES V., VI., & VII.

Cases of Myxœdema (Dr. Wm. M. Ord.)

PLATE V.

FIG. 1. Section of Eyelid: cutaneous surface, showing thickening of fibrous walls of hair follicles and irregular bulging of root sheath, either due to budding from its surface or constriction by surrounding fibrous tissue. Hairs in process of shedding. ($\times 80$.)

FIG. 2. Section of Eyelid; showing meibomian gland, and arterioles with thickened fibrous coat. ($\times 80$.)

PLATE VI.

FIG. 1. Section of kidney; showing great increase of fibrous tissue around some of the Malpighian capsules, and between the tubules; also solid masses, perhaps obliterated arterioles or Malpighian bodies. ($\times 80$.)

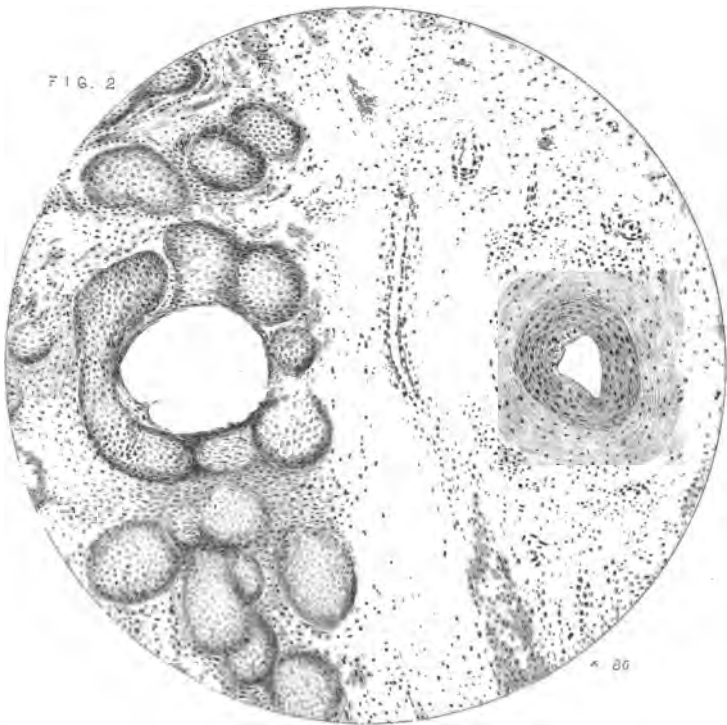
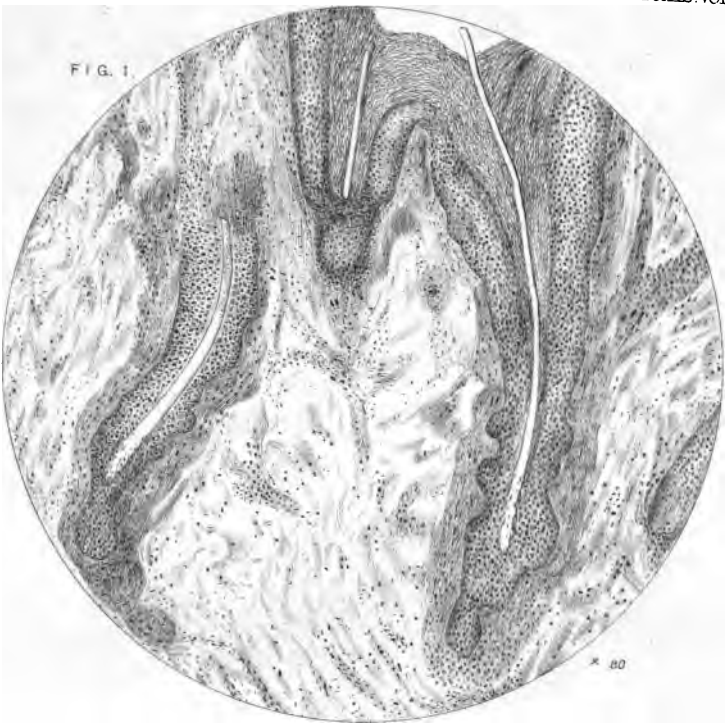
FIG. 2. Section of thyroid body; showing almost complete replacement of its vesicular structure by connective tissue with cell-crowded patches. Fibrous coat of arterioles thickened. ($\times 80$.)

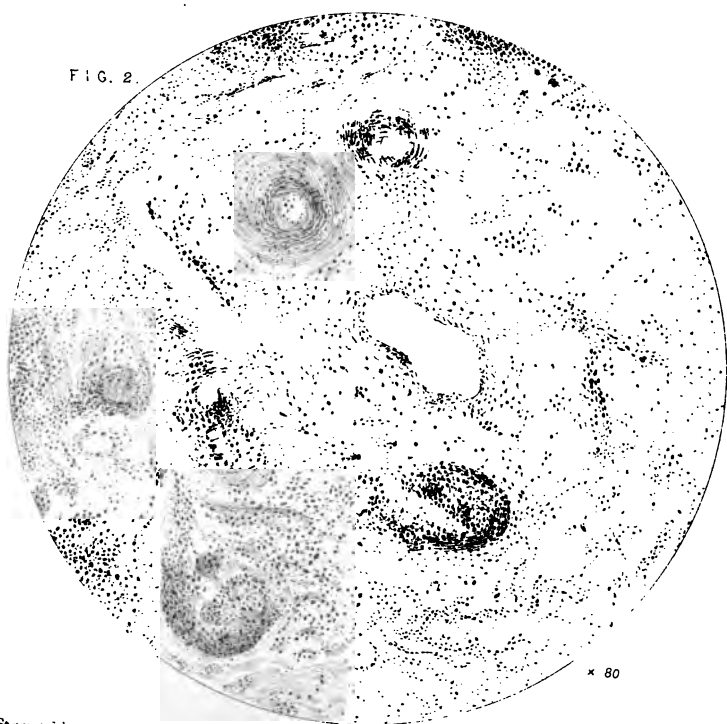
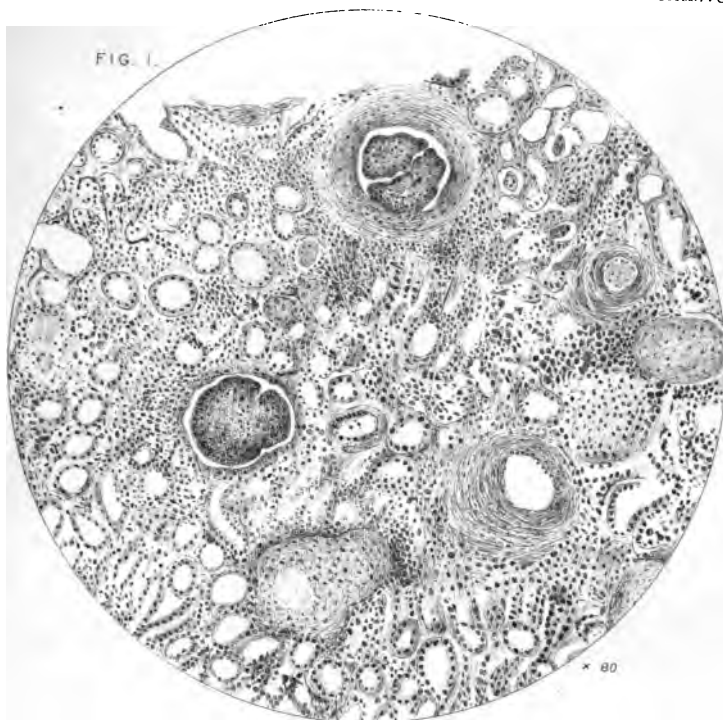
PLATE VII.

Photographs of H—B—, a patient brought for inspection to the Royal Medical and Chirurgical Society on the occasion of the foregoing paper being read.

FIG. 1. Shows the appearance of the patient at the age of 21, before the beginning of the myxœdematous swelling.

FIG. 2. Photograph taken seven years later, shortly before the reading of the paper.







R. Mintern lith.



Mintern Bros imp.

ON THE PATHOLOGY
OF
TETANUS AND HYDROPHOBIA.

BY
JOSEPH COATS, M.D.,
LECTURER ON PATHOLOGY AND PATHOLOGIST
TO THE
WESTERN INFIRMARY, GLASGOW.

Communicated by Dr. W. T. GAIRDNER.

Received December 4th—Read December 11th, 1877.

IN the course of studying the histology of certain of the organs in cases of tetanus and hydrophobia, it has occurred to me that as these two diseases present many points of analogy, as well as many points of difference, they might be profitably studied together with a view to the elucidation of their pathology. It will not be denied that they present many points of analogy if not of similarity. They are both of them commonly related to some injury to the surface of the body. They both present, as their most characteristic features, certain nervous symptoms, of which the more prominent common characters are, a remarkable exaltation of reflex irritability, and the existence of groups of symptoms which may be referred to the medulla oblongata. There is, further, in all cases of hydrophobia, and in many cases of tetanus, a rise in temperature, which sometimes reaches a quite unusual height.

I shall, in the first place, describe the pathological conditions which I have found in the organs examined, and then proceed to discuss the bearing of the facts obtained on the pathology of these diseases.

As to the material at my command, it is, in the case of tetanus, certain parts of the nervous system in five human cases of traumatic tetanus, and one case in the horse. In the case of hydrophobia I have examined the nervous system in two human cases and in one dog, and some of the internal organs in two dogs, the nervous system not being preserved in one of them. The structures were hardened in alcohol and a solution of chromic acid, and thin sections made with the razor. Sections were invariably examined in glycerine, but duplicates were usually coloured with carmine and mounted in dammar or Canada balsam, according to Lockhart Clarke's method. The two sets of preparations could therefore be used for comparison and correction.

I. The Nervous System in Tetanus.

The changes in the nervous system in this disease have been studied by several observers in this country and on the Continent, but we owe our knowledge of the condition of the spinal cord chiefly to Lockhart Clarke, Dickinson, and Clifford Allbutt.¹

The spinal cord.—As to the condition of the spinal cord, I am in substantial agreement with the authors just mentioned. I find in my cases that every region of the cord presents, more or less, the following lesions:

1. There is great overfilling and distension of the blood-vessels, not uniformly, but at intervals. As the larger or medium sized nutritive vessels are situated mostly in the central parts of the cord and in the fissures, it is here, chiefly, that this condition is visible.

2. The most marked lesion is a granular condition around the vessels. This exists more or less in the neighbourhood

¹ 'Med.-Chir. Trans.,' xlviii and li, and 'Path. Trans.' xxii.

of nearly all the larger and moderately-sized vessels; and, as these are situated mostly in the central parts of the grey substance, and in the fissures, it is chiefly met with in these localities, although not confined to them. The characters of this lesion are best seen in specimens which have been mounted in glycerine. In these it is seen that the neighbourhood of the vessel is occupied by a mass of pretty coarse granules. This condition is shown in fig. 1, and it will be seen that in addition to the granules, there are a number of much larger bodies of various sizes and mostly roundish in shape. These are bounded by a well-marked double contour, and are probably drops of myeline, as I have only met with them where the vessel was related to the white substance of the cord, chiefly in connection with the vessels of the fissures. It is a matter of dispute whether this granular material is an exudation or the result of disintegration of the nervous tissue. The appearances presented, especially in glycerine preparations, and shown in Plate VIII, fig. 1, are certainly very suggestive of an exudation; it often looks as if there were a kind of pool in the neighbourhood of the vessel, as if the material had been fluid in the living body, and these little pools sometimes run off to some little distance from the vessels. This view is confirmed by the occurrence of the granular material in the neighbourhood of the vessels which sometimes run transversely in the fissures; here there is sometimes a considerable gap between the granular material which sticks by the vessel and the neighbouring white columns. No doubt there is disintegration of nervous tissue, and the round bodies already mentioned and figured are most likely the myeline from nerve fibres which have been destroyed.

3. There is an appearance which hardly deserves to be separated from the last as a distinct lesion. In many of the sections the blood-vessels and surrounding granular material have fallen out, so that it is quite common to meet with gaps. These are usually round, but sometimes their outline is like the segments of two circles which have met, and it is as if two neighbouring lesions had coalesced. The gaps,

like the lesions they represent, are mostly in the neighbourhood of the central canal, that is to say, to either side of it in the grey commissure.

In one case I have met with a peculiar appearance in the white columns, to which, as it is isolated, I do not attach much importance. It is as if a cylindrical portion of white substance had become necrosed. In transverse sections there is a circular area, in which the nerve fibres and connective tissue do not take on the carmine, and this area is so loosely connected with the neighbouring white substance that in most sections it has dropped out. Of the many sections made, it only occurred in one set, but in these it was found continuous for some distance in the length of the cord.

These lesions were present in the horse as well as in man, although in the horse they were not so pronounced as in man. In the horse, only the medulla oblongata and upper cervical cord were obtained, but these parts were essentially in the same condition as those in the human cases. In the horse the central canal was filled with granular material, and apparently much distended. In the human cases the epithelium of the central canal seemed often unusually abundant; but as I have found this so often under a variety of circumstances, I attach no special importance to it. The most marked example of so-called proliferation of the epithelium of the central canal which I possess is in a cord, which was hardened in order to give sections of the normal cord.

Medulla oblongata.—Lockhart Clarke did not find any lesion in the medulla oblongata in his original case, and Dickinson did not examine it microscopically. In all my cases the medulla oblongata was examined, and I have found it no less affected than the spinal cord. The three lesions described above were all present, though they may have varied in degree in different parts. In addition to these three lesions there were in this part pretty frequent small hæmorrhages, in which the blood corpuscles were frequently insinuated among the nervous structures. These were

not met with in the cord, although possibly they may have been present.

There is in the medulla oblongata a certain tendency to localisation of the lesions, to which I am inclined to attach considerable importance, as we shall see further on. We saw that in the cord the larger and moderately-sized vessels run chiefly in the grey commissure and the fissures, and that the lesions exist chiefly there. In the medulla oblongata one of the most important nutrient vessels runs longitudinally in the posterior parts of the cord, to either side of and slightly behind the central canal or the deepest part of the floor of the fourth ventricle. In nearly every section this vessel is affected, and often there is hæmorrhage from it. It is well known that the nuclei of certain of the cerebral nerves are situated in this neighbourhood. The hypoglossal nucleus is the most prominent and easily recognised of these, and it is remarkable how often this nucleus and the blood-vessel with its lesion are visible in the same field of the microscope. It is not to be understood that the lesions were not present in other regions of the medulla. I have frequently met with gaps in the olivary body and elsewhere, but they are not nearly so common as in the region specially indicated.

The *pons Varolii* was only examined in one case, and it was found that though the above lesions were present, they were much less abundant than in the cord and medulla oblongata. The most aggravated hæmorrhage which I met with was in the pons, and there were evidences of irritation produced by the blood; that is to say, around the hæmorrhage and partly in it, there were large numbers of round cells, which are much more deeply stained with carmine than the red corpuscles or nervous tissue.

Corpora quadrigemina.—These ganglia were only examined in one case, but the characteristic lesions were very abundantly present, at least in one region. The appearances presented are well seen in a transverse section under a very low power. In such a section, made through the testes, there are seen to be several large gaps on either side of

and behind, or above, the aqueduct. Here again it will be seen that the lesion follows the longitudinal vessels.

The *corpus striatum* was only examined in one case. There were a few gaps, but they were very infrequent, and the ganglion seemed to be involved in a very minor degree.

Convulsions.—These were examined in the last two cases which came under observation, and the parts chosen were the motor regions, as determined by Ferrier and Hitzig. In one of the cases the granular condition around the vessels was unmistakeably present but to a very slight extent. This was not found, however, in the other case. In both, there was a lesion which was not observed in the other regions of the nervous system, but which to a certain extent resembles the principal lesion found there. It consists of the exudation of a homogeneous yellow material outside many of the smaller blood-vessels (see Plate VIII, figs. 2, 3, 4, and 5). This condition was by no means homogeneously distributed, there being some parts of the convolutions where it did not seem to be present at all, and others in which it appeared to be present in connection with almost all the smaller vessels. It was only found in the grey matter of the convolutions, and in connection with vessels which were little if at all above capillary size. The exudation occurred in the form of little oval drops, and these retained their yellow colour in sections stained with carmine and mounted in the usual way in Canada balsam. I am aware that great care is necessary in interpreting appearances related to the vessels in the cortex of the brain, and my observations, so far as they have gone, confirm those of Obersteiner,¹ who has shown that in what are regarded as normal brains collections of pigment exist immediately outside the vessels. Even in the brains under review it is very easy to find numerous examples of this aggregation of pigment around the vessels. But the lesion I have described and figured presents several points of difference from these. It occurs mostly in connection with the smaller vessels, while the ordinary pigmentary aggregations are commonly met with

¹ Stricker's 'Jahrbücher,' 1877, p. 381.

on the surface of vessels of larger size than these. Then the ordinary pigment is granular, or contains granules, and lies in an elongated form along the outside of the vessel not apparently compressing it. The other lesion consists of a homogeneous material, having an appearance strongly suggestive of its being formed of a drop of some fluid; it has an oval form, and *in nearly every case it compresses the vessel more or less*, in many cases seriously compromising its lumen. It was this which first attracted my attention, and it is exceedingly striking to find, within a limited area, quite a number of vessels occluded more or less completely, in the way exhibited in figs. 2, 3, 4, and 5.

Nerve.—The nerve proceeding from the injured part was only examined in one case, but no lesion was detected. There was no interstitial neuritis, such as some observers have found.

II. *The conditions met with in hydrophobia.*

As already mentioned I have obtained certain parts from two cases in man and two in the dog. The cases in man have been so fully reported in their clinical aspects by Drs. Dunlop and Patterson in the 'Lancet' of January 27th and February 3rd, 1877, that I need not refer further to them here. The cases were typical ones of hydrophobia.

It was with some difficulty that I obtained possession of the body of a dog which was said to have been affected with rabies, and only after it had been buried for a week. The symptoms in this dog were very carefully observed by Mr. Alexander Henderson, who has considerable experience of dog ailments, and he has kindly given me a note of the facts observed. The symptoms are so well described, and the case seems such a typical one of rabies, that I shall here reproduce the account of them in his own words.

"On Thursday, 23rd November, 1876, I was called to see
s of age, which had returned home after

an absence of four days in a state of great emaciation. Before going off it had been in its ordinary condition, which was that of a healthy dog but not fat. The owner of the dog thought that during its wanderings it must have been ill used. I found the dog lying on a sofa, and on my entrance, instead of, as usual, coming to meet me, he continued to rest. Its master said he could not understand its conduct. On being spoken to by its master, it gave no sign of recognition, but, on the contrary, snapped at him, then, all at once, would run to him, jump on his knee, and then as suddenly return to its sofa. The animal was a poodle of an extremely friendly temper, indeed an extraordinarily even temper. After noticing it for a short time I sat down on a stool and gave the dog my hand to smell, and when he had seen that there was nothing to fear from it, I laid hold of and muzzled him. The dog was very thin, extremely so; and after a careful examination, the only mark I found on him was a little wound about a quarter of an inch below the left eye. Water, soup, and biscuits had been offered, and were placed before him, but he would have none of them. The animal showed a great dislike to light, and seemed to wish to be left alone in the dark. The following morning, Friday, I was sent for, and on arriving I found the beast very much excited; through the night he had torn to pieces the sofa cover, and 'mouthed' the furniture in the room. It was some time before I could touch him; he was quite changed, and though seemingly desirous of being alone, betrayed a constant watchfulness quite different to a dog wishing rest and quiet. He seemed very feverish and ill. I considered it unsafe to allow him to remain in the house, so removed him to a wooden outhouse, where I arranged a warm bed for him in the centre of the floor near the window. The animal, however, preferred to lie on the bare stones in a dark corner to having a comfortable bed in the light. I did not see him again till next morning, Saturday, when I found him much worse. He had still refused meat and drink although showing no dread of water, and at intervals he would get furious, crying and biting at the woodwork in an

unreasoning manner, until utterly worn out he would drop on his side. The floor was covered with small pieces of wood, which in his rage he had torn from the side of the house. There was an absence of foam at the mouth, such as is met with in epileptic fits in the dog; but in this case a small quantity of thick adhesive saliva clung to the corners of the mouth, and seemed to worry the dog. Whether from this cause or not the animal was frequently fighting with his paws at the corners of his mouth, the action being like that of a dog with a bone stuck in its throat, I thought it unsafe to allow him to live, so on Saturday afternoon I shot him. I consider this a genuine case of rabies or hydrophobia in the dog."

Of this dog I preserved the brain and spinal cord, the salivary glands, liver, kidneys, and spleen, and hardened them in alcohol and chromic acid. The other dog was seen during life by Dr. Klein, and he states that it was without doubt a case of rabies. The parts examined in it were the salivary glands, liver, spleen, kidneys, intestines, larynx, and trachea. I shall now proceed to describe the appearances presented in these various cases, and it is to be understood that, unless mention is made to the contrary, the appearances described appertained to the organs of all the cases which were examined.

The nervous system was examined in the two human cases, and in dog No. 1, and in all three the principal lesions were identical.

The principal lesion concerned the blood-vessels primarily, although its effects were not absolutely limited to them. Figs. 6 and 7 show the essential features of this lesion, and it will be seen that around the vessels there is a collection of round cells or leucocytes. In some cases there are only a few lying in the perivascular space, but in others they form a thick mantle, in which the vessel is completely buried beneath layers twenty or thirty deep. This condition does not affect the vessels uniformly; there is no continuous clothing of the vessels with leucocytes, but there appear to alternating with spaces where the condition

described is present. On the other hand, the lesion appears in some cases to occupy a considerable length of the vessel, as it is found that when a vessel happens to be exposed longitudinally, it is often seen to be clothed for a considerable distance with layers of leucocytes. In the dog's nervous system the vessels have often been torn out for some length in making the section, as the tissue was very brittle, and in these sections considerable lengths of blood-vessel are sometimes seen, clothed in a mantle of cells.

This condition is very abundantly present in the spinal cord and medulla oblongata. I am not convinced that any one region of the cord is more affected than another, but perhaps the dorsal region is less involved than the cervical. As the larger and medium-sized vessels of the cord run mostly in its central parts, and as it is chiefly these which are affected, we find that the lesion is most obvious in the grey commissure to either side of the central canal, but it is not confined to these parts, and may even be seen in the white substance. In the medulla oblongata it is present to a very marked extent, and indeed the most extreme aggregations of leucocytes which I have met with have been mostly here. In fact, it looks as if few of the vessels in the medulla oblongata had escaped. There does not seem to me to be so much localisation of the lesion in the posterior parts of the medulla as I found in tetanus, although to a certain extent a similar distribution is manifest.

In the pons Varolii the lesion is also present, and sometimes to a very marked degree, but it does not seem to be so frequent or so aggravated as in the medulla oblongata. In the corpora quadrigemina it was unequivocally present, but distinctly less frequent than even in the pons. The corpus striatum was, unfortunately, not preserved.

The convolutions were examined in one of the human cases and in the dog. There was here a marked contrast with the cord and medulla in respect that the larger and medium-sized vessels were nearly all unaffected, while in the case of those of nearly capillary size it was very common to meet with collections of leucocytes around them, and in



some cases the vessel seemed to be buried in them and compressed.

It is here right to state that Benedikt, in a paper in Virchow's 'Archiv.' vol. lxiv, has, to a certain extent, anticipated these observations. He describes in the brains of dogs which had been affected with rabies, and in one human case, granular disintegration, an exudation of a hyaloid substance, and a kind of miliary abscess in connection with the vessels. The two first of these conditions I have not observed, but the condition just described may be taken to represent Benedikt's miliary abscesses. It is remarkable that, so far as I can discover, Benedikt says nothing as to the appearances in the cord and medulla oblongata, though it is there that I have found the lesion most pronounced.

Besides this lesion, which is the most constant and most noticeable in the central nervous system, there is a condition which seems closely related to it. The superabundant leucocytes are not confined to the neighbourhood of the blood-vessels, but in many parts of the nervous system they were seen to be present in unusual numbers in the tissue generally. The convolutions of the dog especially looked as if infiltrated with round cells. In some parts of the cord and elsewhere the leucocytes were aggregated around the ganglion cells, and occasionally they appeared to occupy the peri-ganglionic space in considerable numbers, as seen in Plate IX, fig. 10. It must be added that this latter is by no means a constant, or, indeed, a very frequent appearance.

The only other prominent abnormal condition observed in the central nervous system was an occasional great excess of amyloid bodies, chiefly near the surface of the corpora quadrigemina. As this, however, is not an unusual condition in other cases, I do not know that much importance is to be attached to it.

It may be added that I have not found any obvious hæmorrhages or areas of granular disintegration. I do not they do not occur, but I have seen no

unequivocal evidences of them, such as I met with in tetanus.

The salivary glands excited most interest next to the nervous system. They were not preserved in the human cases, but in the two dogs they were examined, and in both the same conditions were observed. The glands were everywhere infiltrated with multitudes of round cells (see fig. 11), but the infiltration was greater at some parts than others. They were insinuated in large numbers between the proper glandular structures, but they had accumulated especially in the spaces occupied by the ducts and vessels. In these spaces their numbers are frequently so great as to obscure the outline of the vessels, which appear, as it were, buried in them. It is difficult to make out what relation these cells bear to the vessels, chiefly because the veins and arteries, except the largest, are mostly empty, or, at least, their contents are difficult to make out. It is easy to distinguish the arteries from the thickness of their coats, but the veins are mostly collapsed and buried in the aggregations of round cells, so that the walls are, for the most part, invisible. After careful investigation, however, it can be said that in some places, at least, there is a special accumulation outside of, and along the veins, and even where this cannot so definitely be made out, it is indicated by the existence of elongated groups of cells, the great excess of round cells in the spaces occupied by the vessels being another fact pointing in the same direction.

It may be added that the pancreas was examined in one of the dogs, but no such lesion was found.

The *kidneys* were examined in both dogs. The most obvious condition here is a very marked but not uniform hyperæmia, existing both in the cortex and pyramids, but especially manifest in the veins which run between the cortex and pyramids. So great is the distension in some of these veins that they occasionally look like large sinuses. It is sometimes evident that in the distended veins the white blood-corpuscles are abnormally abundant. This is very manifest in the specimen from which Plate IX, fig. 12, is

taken, in which layers of white blood-corpuscles lie along the wall, sometimes in several layers. There is no extensive extravasation of the white corpuscles, that is to say, there is no infiltration of the kidneys with leucocytes, such as we have found in the salivary glands. In addition to this hyperæmia, which, it should be remarked, is by no means general, but appears localised to certain parts, there are, in one of the cases, frequent hæmorrhages. These are in the form of small collections of blood, and have their seat chiefly, if not entirely, in the cortical substance. They are often pretty close together, but always of small size.

The *liver* was examined in both the dogs. In one there is a general interstitial hepatitis, evidenced by an increase of connective tissue in the course of the portal vessels—in fact, an early cirrhosis hepatis. This is, obviously, a chronic condition, and has nothing to do with the hydrophobia; it was absent in the other dog. Otherwise the livers were hyperæmic, but not very markedly so, and if there is any excess of leucocytes in the connective tissue it is very slight.

The *mucous membrane* of the *larynx* was examined in one of the dogs, and it was apparent that the mucous glands were infiltrated with leucocytes in a similar way to the salivary glands, but to a much less degree. The infiltration was nearly uniform, each acinus appearing as if surrounded by a layer of round cells.

The *cicatrix* of the dog-bite, and the *nerve-branches* for some distance up from it, were examined in one of the men. In the neighbourhood of the cicatrix there were evidences of irritation in the form of leucocytes which infiltrated the skin and subcutaneous tissue. The blood-vessels in the neighbourhood contained, in some cases, a granular material, which was evidently disintegrated blood-corpuscles. In other cases the vessels were partially occupied by transparent, globular bodies, which are often yellow in colour, and are, I believe, red blood-corpuscles altered in some way. This alteration of the red corpuscles is met with in inflamed structures, and may also be taken as an evidence of irritation.

Such being the facts observed, it remains now to consider briefly the bearing which they may have on the two diseases before us. In the first place, as to hydrophobia, we found in the central nervous system certain lesions which I do not presume to regard as distinctive of that disease, but which may, I think, be fairly considered evidences of irritation. There were leucocytes aggregated around the vessels and infiltrated into the nervous tissue often in great numbers. But these evidences of irritation did not exist only in the nervous system. The salivary glands, in the cases in which these were investigated, presented appearances strictly analogous to those in the nervous system—leucocytes around the vessels and infiltrated between the glandular elements. The kidneys also presented changes, the vessels were much dilated, and, more important than this, there were immense aggregations of leucocytes inside some of the veins, and also hæmorrhages. The existence of signs of irritation in all these organs, and the direct connection of these signs with the blood-vessels, is exceedingly suggestive of the existence of some irritant in the blood which has acted on the vessels primarily.

In the cases of tetanus subjected to examination, there were in the nervous system appearances which, though by no means identical with those in hydrophobia, are still to my mind strongly suggestive of irritation, and of irritation acting out from the blood-vessels. There is an exudation around the vessels, or a granular disintegration, and this lesion certainly suggests some irritant within the vessels. It is also to be noted that, while the relation to the blood-vessels of the respective lesions in tetanus and hydrophobia is similar, the distribution of these lesions in different parts of the nervous system is also remarkably similar. In both the lesions occur around the larger and medium-sized vessels of the cord, around the vessels of the medulla oblongata in a high degree, and in the convolutions to a slight extent. In tetanus the other organs have not been examined, and it cannot be said whether there are evidences of irritation in them.

In regard to the symptoms in these two diseases, few will deny that in hydrophobia they are related to some poison circulating in the blood, and attacking specially the central nervous system. In the case of tetanus this view, though supported by very high authorities, is not generally received. Looking, however, to the fact, that on the one hand the symptoms in both these diseases have a closely analogous anatomical distribution, and that on the other hand the lesions though different in kind are so similar in distribution, it seems to me very natural to suppose that in tetanus also there may be some poison circulating in the blood and causing disturbance. In this connection it may be said that the high temperatures observed in tetanus, sometimes reaching a startling elevation, are more suggestive of a general disease, these temperatures not bearing any constant relation to the exaggerated muscular contraction.



DESCRIPTION OF PLATES VIII and IX.

Pathology of Tetanus and Hydrophobia (Dr. Jos. Coats).

PLATE VIII.

FIG. 1. Tetanus. From a section of the medulla oblong. at its lower part. A collection of granular material is seen, in which are larger bodies, probably drops of myeline. (Hartn. vii, 3 = 250 diams.)

FIGS. 2—5. Tetanus. From the convolutions. Oval drops of a yellow material are visible outside the vessels, often compressing them. (Hartn. vii, 3 = 250 diams.)

FIG. 6. Hydrophobia. From a section of the spinal cord. Aggregation of leucocytes around a vessel. (Hartn. vii, 3 = 250.)

PLATE IX.

FIG. 7. Hydrophobia. From a section of the med. obl. at the lower part of the fourth ventricle. A vessel as if clothed with leucocytes. (Hartn. vii, 3 = 250.)

FIGS. 8, 9. Hydrophobia. From the middle of the cervical region of the cord. Leucocytes around ganglion cells.

FIG. 10. Hydrophobia. From the middle of the cervical region. Leucocytes around ganglion cells. (Hartn. ix, 3 = 460.)

FIG. 11. Rabies in dog. Section of parotid gland, showing leucocytes infiltrated between the glandular acini. (Hartn. vii, 3 = 250.)

FIG. 12. Rabies in dog. Section of kidney. A vein highly distended, and with great aggregation of white corpuscles. (Hartn. iv, 3, with draw-tube = 120.)

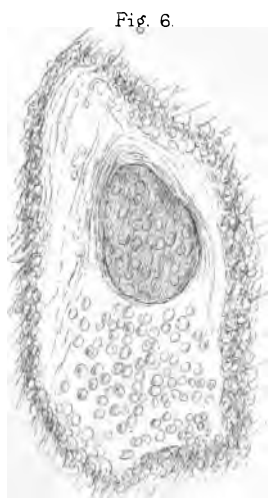
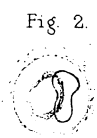
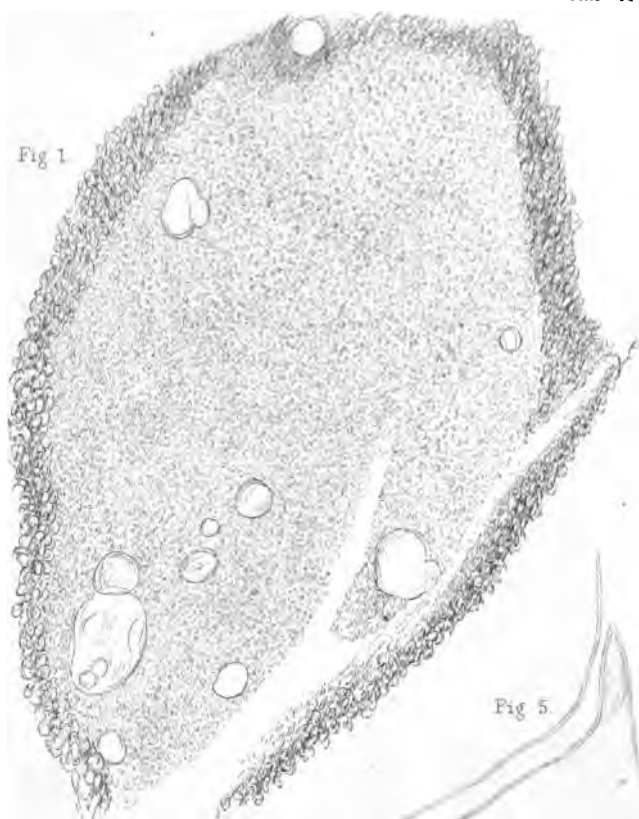


FIG. 7.

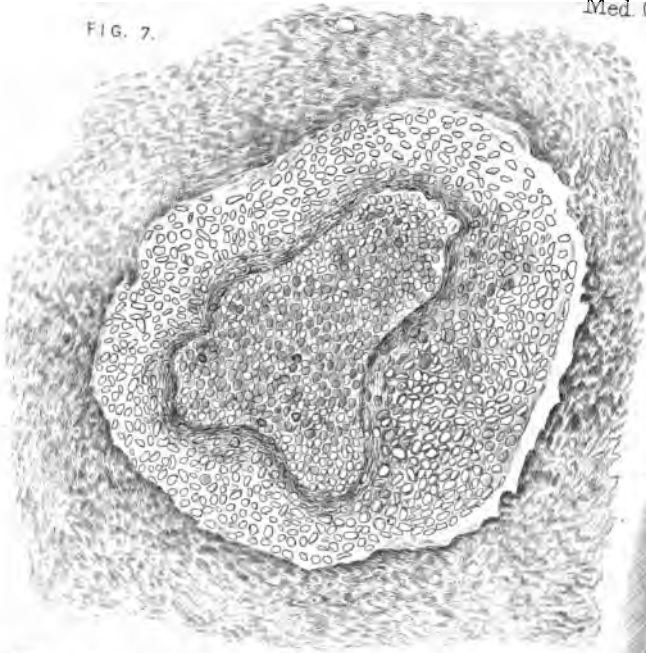


FIG. 9.



FIG. 8.

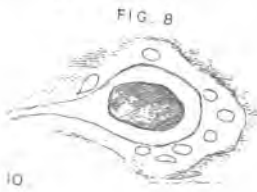


FIG. 10.

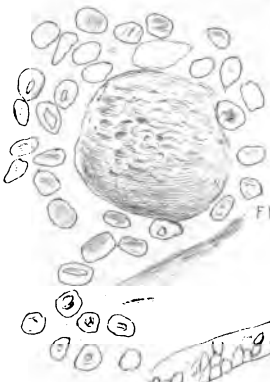
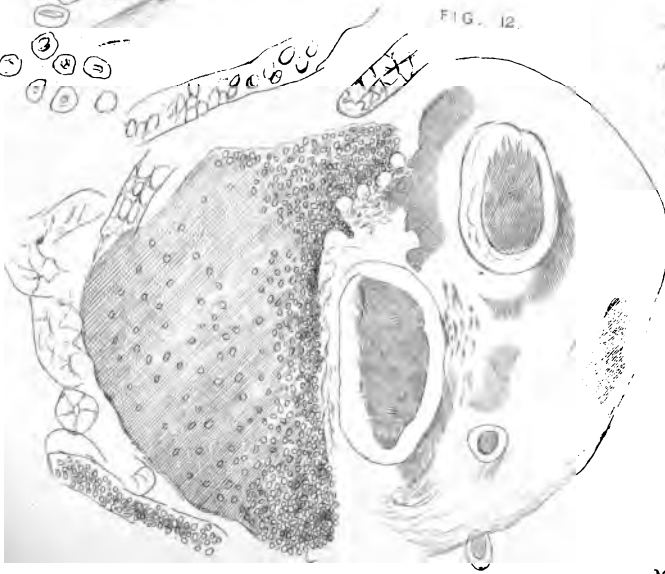
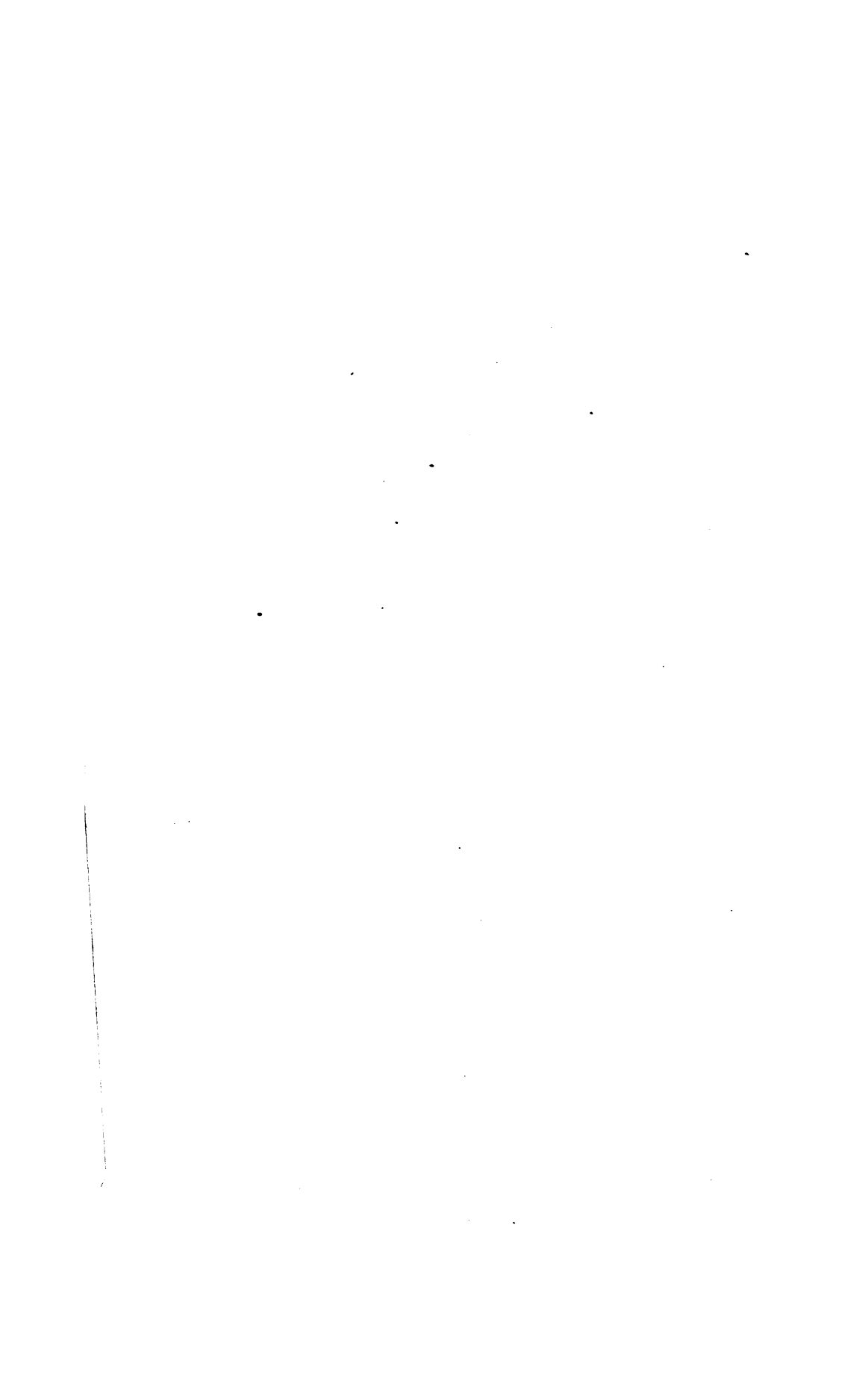


FIG. 11.



FIG. 12.





ON THE
PROPORTION OF RED CORPUSCLES IN
THE BLOOD

IN
SOME SKIN DISEASES.

BY
GEORGE THIN, M.D.

(Received November 24th, 1877.—Read January 22nd, 1878.)

THE enumerations of the proportion of red corpuscles in the blood recorded in this paper were made by the method and instruments recommended by M. Malassez, of Paris. In order to obtain a basis for comparison, the blood of healthy adult persons and of persons suffering from exhausting diseases was examined with the following results:

1. *Healthy persons.*

MALES.	Red corpuscles per millimeter cube.
Butcher's assistant, æt. 22	4,760,000
Greengrocer's assistant, æt. 28	4,700,000
Baker's assistant, æt. 22	4,600,000
Physician, æt. 26	4,400,000

FEMALES.				Red corpuscles per millimeter cube.
Domestic servant, æt. 29	.	.	.	3,880,000
„ „ æt. 31	.	.	.	4,690,000
„ „ æt. 24	.	.	.	4,260,000
„ „ æt. 19	.	.	.	4,020,000
Girl, æt. 13	.	.	.	3,940,000
Married woman, æt. 25	.	.	.	4,210,000

II. *Persons suffering from exhausting diseases.*

Man, æt. 58, with obstruction of the œsophagus	3,500,000
Young woman, very markedly anæmic	3,110,000
Man, æt. 29, in advanced phthisis ¹	2,160,000

The numbers given in the Table appended to this paper were found in persons affected with skin diseases, the cases being selected from patients in whom the condition of the blood was not changed by the coexistence of other morbid conditions, or by the exhibition of drugs.

An examination of the results obtained in the six cases of eczema shows that in only one of them (an old man, æt. 60) was the number of corpuscles below the average, whilst in two the number was unusually high. The cases were selected as being likely to test whether a diminution of the red corpuscles is associated with eczema, embracing, as they do, instances in which the disease was recent and spontaneous, and others in which it was chronic and inveterate.

On reviewing the facts as a whole it will be seen

¹ These figures are in general accordance with those published by M. Malassez in the *Comptes Rendus de la Société de Biologie* for 1874, and in the *Bulletins de la Société Anatomique de Paris* for 1874. According to M. Malassez's observations 4,000,000 is a low number, and anything over 5,000,000 a high number for a healthy adult man. In females the proportion is less than in males. In his observations on persons suffering from cancer the maxima numbers in men are 3,800,000, the minima 2,500,000; in women, maxima 3,080,000, minima 2,560,000. In phthisis, his maxima numbers for men are 4,480,000, minima 2,560,000; for women, maxima 3,900,000, minima 980,000.

that the only cases of skin disease in which the corpuscular element of the blood was found deficient were a case of chronic pemphigus and a case of prurigo. On the other hand it is clear that psoriasis, eczema, and leprosy develop in persons in whom the number of corpuscles is normal. This is especially noteworthy as regards eczema, a disease in which theoretical notions regarding the blood have influenced treatment to a considerable extent.

The author records with pleasure his thanks to Mr. Morrant Baker, Dr. Cayley, Dr. Tilbury Fox, and Dr. Payne, for their kindness in assisting him to procure suitable cases, and for the interest they have taken in the inquiry.

Sex.	Age.	Disease.	No. of red blood-corpuses per millimeter cube.	Remarks.
M.	25	Psoriasis	5,250,000	Nummular form thickly scattered over trunk and limbs. Had had several previous attacks.
M.	25	Psoriasis	4,820,000	Frequent attacks during the previous 15 years.
F.	27	Psoriasis	4,330,000	Eruption copious. Suckling her infant.
F.	18	Psoriasis	4,448,000	Ps. guttata. First attack.
M.	60	Eczema	3,750,000	Universal chronic eczema for 4 years. Been worse during the previous six months. Had had a severe attack of eczema at the age of 25.
M.	19	Eczema	4,200,000	Universal chronic eczema lasting 8 years. The epidermis desquamating copiously from a congested cutis over nearly the whole body.
F.	12	Eczema	4,420,000	Scaling eczema of arms, and eczema rimosum of lips. Had had previous attacks.
F.	26	Eczema	4,350,000	Dry eczema of scalp, flexures of arms, and on several parts of the trunk.
F.	20	Eczema	3,880,000	Impetiginous eczema of the cheeks and neck.
F.	25	Eczema	5,050,000	Acute eczema of the whole skin of both hands and wrists. Second attack within a month.
M.	30-40	Pemphigus	3,580,000	Chronic.

F.	16	Acne Sebacea	4,050,000	Very numerous colourless papules on forehead, cheeks, and chin, each papule filled with a sebaceous plug. No inflammatory base.
M.	13	Prurigo (Hebra)*	3,420,000, after interval of 1 month's treatment 3,550,000	The disease began in infancy, and had continued without intermission.
M.	47	Acne Rosacea	4,140,000	Second stage of the malady. Skin on nose and cheeks much thickened. The patient had been long in a semi-tropical climate.
M.	17	Leprosy (anæsthetic)	4,320,000 4,470,000 4,170,000 4,330,000	On March 1st, 1877. On March 26th, 1877. On April 1st, 1877. On May 26th, 1877. During this period the patient was taking iron. There was no improvement in the disease. The proportion of white corpuscles to red was valued once, and found to be 1 to 50. The disease was not in an advanced stage.

* As this disease is hardly recognised out of German-speaking countries, and as the term prurigo is often applied in England to affections of a totally different nature to that alluded to here, it is necessary to state that this patient's malady is a well-defined example of the affection described by Hebra. During the twelve months that the patient has been under the author's observation the behaviour of the eruption under treatment has been the usual one—speedy alleviation and constantly recurring relapses. The boy was born in Australia. In a younger brother, aged seven, also born in Australia, and subjected to the same hygienic influences, the number of the red corpuscles was 3,970,000.

And

A SECOND COMMUNICATION
ON
SIMPLE ATROPHIC SCLEREMA.

BY
JOHN HARLEY, M.D. LOND.,
SENIOR ASSISTANT PHYSICIAN TO, AND LECTURER ON GENERAL ANATOMY
AND PHYSIOLOGY AT, ST. THOMAS'S HOSPITAL.

(Received December 11th, 1877—Read January 22nd, 1878.)

LOUISA M—, a well-developed, unmarried woman twenty years of age, 5 feet 2 inches high, of fair complexion, with blue eyes and yellowish-brown (light auburn) hair.¹ She was formerly stout² and of a high colour, now she is thin, semianæmic, and, to a great extent helpless, on account of the condition of her hands. Her family history is moderately good. Both parents are living and in good health, the father forty-seven, the mother forty-three, and both have been free from disease and from tendency to rheumatism or dropsy.

Louisa M— is an only child, and her mother was twenty-five years of age at her birth. Her only paternal uncle died of consumption, "brought on by his own indiscretion." Her paternal grandfather died of "typhoid fever" at the age of

¹ The teeth and hair are strong and healthy, as in the former case.

² Her weight at different times is given at p. 106.

forty; her paternal grandmother died of "nervous" debility when over seventy years of age. On her mother's side the grandmother is still living and in good health, and the grandfather died only two years ago. One aunt had rheumatism severely at the age of twelve; she recovered well, but at the age of twenty had a second attack, which completely crippled her, and she was unable to close her hands. She died in her thirtieth year. For five years before her death she was completely blind, and for six months before the final event she was unable to move without assistance. Another aunt, married, but barren, is at the present time attending a hospital for a "tumour of the side." A married cousin (Lydia M—) was bedridden for some years after puberty and could not move without help.¹

To return to Louisa M—. She had variola badly when only a year old. At the age of two and a half years she had a severe attack of "gastric fever," this was doubtless enteric; she was in bed a month and had diarrhœa and bloody stools. Subsequently she had hooping-cough, and about the age of ten a slight attack of measles. As she grew up the health was moderately good. She was unable to bear much excitement, which produced headache; and deviation from plain diet caused indigestion with pain in the epigastrium. The catamenia appeared at the age of fourteen, and the functions were normal up to eighteen months ago, when there was suppression and its results—headache, light-headedness, flushing heats, and pain in the back for six months; since then the flow has been regular, but scanty, and preceded and followed by some leucorrhœa.

In May, 1875, she went from home, and was engaged for about six weeks making ice; her father discovering that she

¹ She died under the care of Dr. Ashurst, of Farningham, who has kindly sent me the following particulars:—"She came under my treatment for arthritic rheumatism described as following an attack of fever. The rheumatism persisted, and was accompanied by inflammatory attacks in the pleuræ, and hepatic congestion; and she ultimately drifted into an asthmatic condition, in which she died. The heart from the first showed great feebleness, but no valvular mischief. She was very corpulent. Her father died of dropsy; her mother of apoplexy."

was living badly removed her to her home after an interval of about four months. Excepting this interval she has always resided at home, where she has been engaged in the work of the house and lived comfortably.

The last few years she has been liable, at intervals of two or three weeks, to attacks of syncope—partial loss of consciousness for some seconds, rushing sound in the head, and deadly pallor, preceded by a flush of heat, and followed by a cold sweat, during which the hands would be quite wet. Some weeks she had had two or three of these attacks.

Two years ago, *i. e.* shortly after her return home, her health began to fail, she became weak, thin, low-spirited, and for a month the fingers were successively affected with sub-epidermic whitlows. After this affection passed off the fingers gradually became cold, stiff, and hard.

In July, 1876, while at the seaside, she had some swelling of the feet, and of one leg as high as the knee. The knee and toes were red and tender, and for two or three days she could not bear movement of the knee nor get her boots on. The whole affection lasted but a week.

The following is her present condition:—The hands are very cold and hard, the fingers semiflexed and almost fixed. They are rather shiny and tense, but are not enlarged, and the integument feels as if it were of the consistence of tendon; it is impossible to pinch together a fold, or to produce a wrinkle. On favorable days the colour is sallowish-pink, but on cold damp days the fingers have a leaden hue. On attempting to close the hands the knuckles become sallowish-white, and after the greatest effort the finger-tips impend over the palm at a minimum distance of an inch. The wrists are maintained straight; flexion and extension are much diminished, but the rotatory movements of the wrist are freer. The wrists ache, and all but the gentlest movements cause pain. A fine creaking is palpable during movement, when a finger is pressed upon an extensor tendon. The finger-tips are attenuated, and the nails strongly incurved from side to side, so as to reduce the interval between the edges of the nails of the little fingers to a quarter of an inch

and to three and a half tenths of an inch in the rest. The nails of the index fingers are not so incurved, but they are coarse from previous suppurative action around them. These features are slightly more marked in the right hand.

The tactile sense is slightly diminished, the fingers cannot anywhere discriminate the two points of the compass when they are separated by an interval of two lines. (This may be in part due to disuse.)

The wrists and lower part of the forearms are always cold.

After sitting for an hour in a warm room of temperature of 58° Fahr., that of the mouth was 98°, of the right fingers 66°, and of the left 69°.

The sclerema decreases upwards, and as a distinct affection ceases just below the wrists. The integument of the forearm, however, is brawny up to the elbows, the muscles are atrophied, and the anterior aspect of the limb is flattened.

The elbow-joint is slightly affected, so that complete extension of the forearm is impossible.

The right hip-joint is stiff and slightly painful, causing her to walk lamely at times. The feet are normal, but just where the ankles escape pressure from the edges of the elastic sides of the boots the integument is firmer than normal. There is no trace of œdema, nor are any of the superficial veins enlarged.

The adipose tissue has entirely disappeared from the neck and upper half of the front of the chest. On the sides of the neck, from the level of the hyoid bone downwards to the lower border of the first rib, the skin presents the characteristic appearance described in my previous case.¹ It is due to the formation of *lineæ atrophicæ*, which take an oblique course downwards and inwards to the root of the neck, and turning over the clavicle and first rib mount upwards over the manubrium to meet those of the opposite side, thus forming a series of semicircles around the root of the neck and the upper limit of the chest in front. The skin itself is thin and pliable, and there is at present no subcutaneous

¹ See 'Medico-Ch'

ration. On twisting the head aside, the lineæ atrophicæ are brought out conspicuously, and apparently in relief, but really this is not so, for the finger cannot detect the least irregularity of surface. The appearance of prominency is due to strong contrast of colour, the lineæ atrophicæ alternating regularly with lines of skin which has not undergone the atrophic change, but which has received an increase of pigment. Thus there is a very regular alternation of lineæ atrophicæ and lineæ pigmentatæ, the former are of ivory whiteness and opacity, the latter are yellowish brown and of the exact tint of the hair of the head. On examination with a pocket lens both kinds of lineæ are seen to be pervaded by a coarse, meagre, network of fine hair-like vessels, the main branches of which run in or close to the borders of the lineæ pigmentatæ. The meshes of the network are oblong and take the direction of the lineæ, the finest branches pass into the lineæ atrophicæ, where they become scant and soon disappear by attenuation. These features are represented in Plate X.

The patient herself thinks the neck has become a little browner of late, and she has noticed that on throwing back the head the skin in front of the neck is very tightly drawn. On doing so the fascia covering the mylohyoid is thrown into three tight thin cords, a central and two lateral, and the pressure upon the larynx excites deglutition.

A few fine blood-vessels are seen by the aid of a lens on the summits of the shoulders, and here and there in the cheeks, but elsewhere (as far as I have examined) they are absent; and on scanning the healthy skin of the shoulders and chest the regular pigmentation of the orifices of the sebaceous follicles arrests attention, for the surface is crowded with regularly distributed yellowish-brown, circular dots, commonly a little raised and surrounded by a very fine, radiated, wrinkling of the white cuticle.

The integument of the face is rather firm, the expression set, and rather sad, features which are not abolished by a smile. I have never seen her features in the least degree fact which I noted in my former case, and

which occurred *pari passu* with the advance of sclerema on the face.

The mammæ are well developed. The lungs and heart normal. Of the latter the action is easily excited, but in a state of rest the pulse is from 70 to 80, and under all circumstances the rhythm is perfect and the beats equal; the systole is weak, and the first sound is rather flapping, as in conditions of debility.

The urine is normal, but poor, the sp. gr. averaging about 1012. Last summer I once found excess of uric acid, and a copious deposit of oxalate of lime, sp. gr. 1016.5.

She is liable to heartburn, and of late she has vomited once or twice a week a little frothy mucus, sometimes sour and sometimes bitter.

The bowels act irregularly; they are usually constipated, but she is liable to diarrhœa. The tongue is clean. She cannot walk more than a mile and a half on account of aching in the hips and legs, pain in the left side, and a feeling of nausea.

When warm in bed, she occasionally suffers considerable aching pain in the hips, wrists, and palms. The condition of the hands renders her very helpless, and she is unable to turn in bed.

She is chilly, and wears a complete investment of flannel. A table is appended, showing the measurements of the body and limbs, and the weight:

Measurements—

Round the waist	21½ inches.
„ centre of upper arm	10½ „
„ „ forearm	7½ „
„ wrist above and below the end of the ulna	7 „
„ last proximal phalanges	2½ to 2¾ „
„ ankle	8 „
„ calf	11½ „

Weight in her ordinary clothes—

Two years ago she weighed nearly 9 stone. Last summer she was a little over this weight. At the present time she weighs 7 st. 10 lbs. 12 oz.

Such is the history of this patient. Standing alone it would appear to have no definite signification, but side by

side with the patient whose history I have recorded in the last volume (vol. lx) of the 'Medico-Chirurgical Transactions,' it is very interesting.

The exact resemblance of the two cases is remarkable. Six years ago William W— was in precisely the same condition as Louisa M—. Both came to me because they were unable to close the hands, and at the same period of the disease these members were in precisely the same condition. Both had acquired a delicacy of constitution, probably from acute disease—in the one case typhus, in the other variola and enteric fever—in early life. Both had a previous tendency to syncope, and both exhibited the same condition of debility, and the same functional derangement of the stomach. Both exhibit a remarkable placidity.

To conclude, they have one complexion in common, and under the influence of the same disease the same sad fixed expression.

Both patients are here to night, and in the demonstration of this singular condition I esteem this a fortunate circumstance. As these are the only instances which have come under my observation, I have hitherto regarded the affection as a rare one, but like many other presumably infrequent diseases it may prove to be more common than I have supposed it to be.

In my former communication I have fully described the central complications of which the sclerema is, as I believe, but the outward expression, I will therefore, in conclusion, direct attention to two points; 1st, and by way of appendix to my former paper, the present condition of the first of my patients; and 2nd, the formation of the *lineæ atrophicæ*.

1. Those who may remember W. W— a year ago will find him but little altered. He is weaker, more emaciated, and the functional disturbance of the heart and alimentary canal is more severe, prostrating attacks of sickness and diarrhoea with severe colicky pains keep him in bed for days together. Although the radial arteries are wide and healthy, the hands have apparently reached the stage when dry gangrene is imminent. Indeed, the tips of some of the fingers are in-

vaded by a more active form of molecular death than that to which they have been previously subjected. It is accompanied by much gnawing pain. In my description of the hands, I omitted to mention the attenuation of the tips of the third, fourth, and fifth fingers, and the strong incurvation of the sides of the nails, corresponding exactly to what is seen in the other case.

2. As to the morbid process in the formation of the *lineæ atrophicæ*. If the neck in the more advanced case of the disease be examined with a lens, it will be seen that the ivory-like *lineæ atrophicæ* are, with the exception of a straggling vessel here and there, entirely destitute of blood-vessels. In the more recent case, however, the whole of the affected area is pervaded by a network of fine arteries, while no such vessels are seen in the healthy skin. It would appear then at first sight, that in the production of the *lineæ atrophicæ* there is an excessive vascular supply. The reverse of this is really the case: the process is one of atrophy, pure and simple from the first; there is no antecedent congestion, no hypertrophy.¹ By shrinking of the *lacunæ*—canalicular system and blood-vessels of the connective tissue of the corium and subjacent areolar tissue, it passes into a close, firm, bloodless tendinous structure, which by preventing the flow of blood at frequent intervals causes for a time dilatation of the blood-vessels in the alternate lines or areolæ of healthy skin. These latter receive an increase of pigment just as occurs in the delicate skin of some patients after the application of a sinapism.

The condition depicted in the accompanying illustration is therefore indicative of the early stage of the process. The streaky vessels there represented are really on their way to extinction, and when this occurs the pearl-like *lineæ atrophicæ* will stand out in greater contrast to the *lineæ pigmentatæ*.

Appendix, July, 1878.—Both patients are still in attendance at the hospital; the younger, L. M—, is but little

¹ The part was never the seat of irritation, on the contrary, there was diminution both of tactile sensibility and of temperature throughout the process.

altered. She is a trifle more helpless, thinner, and the disease is more marked in the face, the integument having become firmer, and the expression more set. Stiffness and some pain on movement have affected the other hip. The inner ends of the clavicles once were a little tender, and they appear to be somewhat enlarged. Once also she complained of a little aching at the root of the thumb and in the middle finger. These pains appear to be due to stiffness of the joints, rather than to a distinct rheumatismal affection. On three or four occasions she has had an attack of retching or vomiting, but only once a slight return of syncope. I have observed that she is unable to protrude the tongue, the strongest efforts only placing the recurved tip in contact with the retracted lower lip. She is not aware of any previous inability to protrude the tongue, and its forward movement is probably restricted now by sclerema of the connective tissue near it.

William W— is now 51 years of age. He weighs without his clothes only 7 st. 11 lbs., the adipose tissue has everywhere disappeared, and the following measurements indicate the degree of emaciation:—round the middle of upper arm $7\frac{1}{4}$ inches, round the calf $10\frac{1}{8}$, round the right wrist $6\frac{1}{8}$; the left is $\frac{3}{8}$ in. larger on account of a puffy swelling over the end of the ulna. The intervals of retching and vomiting are shorter, and the attacks are more severe and protracted; some weeks they occur every day. Perspiration is still profuse. The skin over the upper part of the chest remains free and supple.

In August he had a more severe attack of diarrhoea and retching than usual, causing for some days alarming prostration, and the hands were quite black. He rallied, however, and in the course of nine days was able to leave his bed. A fortnight later he had another attack which, proved fatal.

DESCRIPTION OF PLATE X.

Simple atrophic sclerema (Dr. John Harley).

Process of cutaneous atrophy in a case of simple atrophic sclerema. Louisa M—, æt. 20. The brown streaks represent the natural skin with an increase of pigment : the intervals, ivory-like areolæ or *lineæ atrophicæ*. Both are pervaded by a coarse network of fine hair-like vessels, the main branches of which run in or near the borders of the *lineæ pigmentatæ*, and forming oblong meshes, spread their finest branches in the *lineæ atrophicæ*, where they become scant and attenuated, and disappear.

The part of skin represented is that which overlies the right clavicle about the centre and the neighbouring integument above and below.



$\times 2\frac{1}{2}$ diam.

AN
ANALYSIS OF SEVENTY-FIVE CASES
OF
"WRITER'S CRAMP" AND IMPAIRED
WRITING POWER.

BY
GEORGE VIVIAN POORE, M.D., F.R.C.P.,
ASSISTANT PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL; PHYSICIAN TO
THE ROYAL INFIRMARY FOR CHILDREN AND WOMEN.

Received December 11th, 1877—Read February 12th, 1878.

WITH one exception (No. 1) all the patients whose cases are tabulated in the accompanying schedule sought my advice for "writer's cramp," or some trouble involving the right hand. In all of them the condition of the hand completely overshadowed any other disease, whether general or local, from which they were at the time suffering.

I have purposely included a few cases of paralysis and spasm of the hand which are obviously not "writer's cramp," because the study of them is calculated to throw some light upon the main subject. As a matter of fact, it may be stated that one of the cases of paralysis had been diagnosed as "writer's cramp," and one of the cases of spasm had been induced solely by overwriting.

I may begin by stating that these cases do not include a single example of that form of loss of writing power (agra-

phia) which is allied to aphasia and is dependent upon lesions of the left cerebral hemisphere. A patient suffering from agraphia is unable, apparently from loss of memory, to portray the forms of the letters by any method, although the various muscular mechanisms which may be employed for writing remain unimpaired. The cases forming the subject of the present paper are all of them examples of failure, from one cause or another, of one of the muscular mechanisms which we have educated to perform the function of writing. In every case the mental potentiality for writing was practically perfect.

The cases are arranged in groups, and in discussing them I shall proceed from the simple to the complex, beginning with those which least resemble writer's cramp and finishing with those which really seem to merit that name.

The *first group* consists of six cases of *paralysis* of the hand, and serves to show to what extent definite nerve lesions involving the hand may interfere with the power of writing.

No. 1 was a draper, æt. 65, who died in August, 1877, in University College Hospital from the bursting of a large right axillo-subclavian aneurism. By its pressure on the brachial plexus the aneurism had produced a paralysis of the right hand and arm which, at the time of death, was absolute. The patient stated that his earliest trouble in connection with his fatal illness was an impairment of writing power, which he had noticed nine months previously. He said, "I could only write by using great effort, and then only illegibly. I was obliged to throw down my pen every half line, and once after carrying a carpet bag in my right hand," which had necessarily increased the pressure on the brachial plexus, "I was quite incapable of writing at all. One of my greatest difficulties was in crossing t's."

No. 2 had complete paralysis of the extensors of his wrist, thumb, and fingers, depending on neuritis of his musculospiral nerve. He was a banker, and impaired writing power was the first symptom to attract his attention, but by the help of an apparatus which kept his wrist extended and

Function of writing.	Tremor.	Neuralgia pain.		Treatment.
of power of	—	—	"I could	Rest, iodide, &c. The man was too weak for surgical interference. Swelling of right wrist.
ty in many	No	—	Special bag in his symptoms	Iodides, galvanism.
the right hand			es, and he	His wrists were swollen, but this disappeared, but reappeared once after drinking port wine. Considers himself decidedly gouty, and has been at Buxton. The thickening of musculo-spinal got quite well.
as of sensa-	"	Yes	ly. Holds	Nil. In <i>statu quo</i> .
him great			in which	
with many				
cts, such as	Yes	No	fibrillary	Iodide of potassium, mercury to salivation. The waters and baths of Aix la Chapelle. Galvanism.
his clothes,			ad all dis-	The general health improved, but the hands remained in <i>statu quo</i> . Swelling observed on back of wrist-joints.
are difficult.	?	None	sensation	Blisters to nerve-trunk, and tonics inter-
age his pen			med—so	nally.
help of his			me a com-	The recovery was complete after some months.
Cannot beat			Some years	
without con-			properly.	
veying the size			and there is	
ple. Cannot				
oves, because				
to wriggle her				
them				
ve without	Of both	—	My friends	
hands. Can-	hands, but			
his shirt well	most of right.			
	Fibrillary			
	tremor of left			
	1st dorsal			
	interosseous			
	Fibrillary			
	tremor of			
	interosseous			
ecup	Marked of	—	een much	
	both hands		the nervous	
			most, it is	Rest; bromide, gentian, and soda; gal-
			re is rheu-	vanism.
			ing of left	His improvement was very marked.
			sleeping.	
			lastic and	
d fine acts	Slight but	Excessive	er suffer-	Absolute rest; iron, bromides, arsenic,
with the right	well marked	after work		quinine, strychnine, belladonna (internally
sted by pain	in right arm	in right arm		and externally), were productive of no
p the arm.				good. Faradism caused pain and made
marked in				her worse. Galvanism and rhythmical
				exercises caused some alleviation. Blisters
				to the nerves and iodides internally
				were productive of marked good, but
				when last seen in June, 1876, an attempt
				to return to work had brought back all
				the symptoms.
d fine acts	?	Excessive	rolonged	Rest, rubbing, douches, blisters round
by pain.		after work,	of a sheet	wrist-joint, iodine paint, were of no
very tremu-		and especi-	for some	service. Improved with belladonna lini-
ment arrange		ally along	perfectly	ment and the galvanic current, and after
ment fingers		the ulnar	under the	a fortnight's treatment wrote 8 sheets of
help of left		border of arm		note-paper and did sewing.
g is as diff-				
ing				

forcibly extended his fingers after flexion, he was enabled to continue writing.

No. 3 had a lesion of the median nerve at the wrist. He wrote with difficulty, and was obliged to watch his pen very narrowly because of the absence of sensation in the thumb and forefinger.

No. 4 had the same motor paralysis as No. 3, with the addition of paralysis of the flexor longus pollicis. He had no sensory paralysis. Being wholly unable to flex his thumb he held his pen by extending his thumb against the index finger. His writing was slowly produced, but not objectively changed.

Nos. 5 and 6 were cases of ulnar paralysis, No. 5 of the entire nerve below the elbow, No. 6 of the deep palmar branch only. The ulnar nerve supplies thirteen and a half of the eighteen intrinsic muscles of the hand, and its integrity is very necessary, more necessary than that of any other nerve of the hand, for all delicate manipulation, especially writing. Neither of these patients were able to arrange the pen between the fingers without the help of the left hand, the interossei being paralysed. The writing was slowly produced and tremulous, so that No. 5 had to retire from a competitive examination in which he was engaged. No. 6 presented features of interest which make it worthy of being given in detail.

The Rev. Mr. R— consulted me in June, 1875, for impaired writing power. He was a public preacher and lecturer, and had previously suffered from nervous exhaustion. Three months ago he had been disturbed at night by a pricking sensation in the hand, and the next morning he wrote with great difficulty. At the time of his visit there was no loss of sensation in the hand, and the muscles of the little finger were acting healthily. There was wasting and paralysis of the interossei and abductor pollicis, and there was characteristic clawing of the fingers. A diagnosis of Cruveilhier's atrophy had been previously made, but the fact that the atrophied muscles all gave the so-called degenerative reactions when tested with electricity made it more probable

that definite nerve lesion was the cause of the trouble. This supposition was confirmed by the discovery of a tender point in the palm near the base of the fifth metacarpal bone, and the eliciting of a history of a bruise at this spot, sustained while moving furniture. The patient completely recovered, but the act of writing is still an effort. He has since suffered from sciatica with consequent wasting of the calf.

The *second group* consists of five cases of obvious *spasm* affecting the right hand and arm. In three of these cases there had been a previous attack of hemiplegia, one was congenital, and one appeared to have been developed by excessive writing, and to have originated as writer's cramp.

No. 7 was a young married lady, æt. 23, who had been troubled with spasm of the right hand since the age of seven, when she had had an attack of hemiplegia following diphtheria. There was marked contraction of the flexors in the right forearm, *and obvious wasting of the extensors. These latter manifested a diminished irritability to faradism.* The spasm was entirely in the direction of flexion, and was most marked in the ulnar muscles. The spasm was aggravated when the hand was called upon to perform delicate acts, and especially in the presence of others. The proximal phalanges were flexed, and the distal extended by an interosseal spasm, and the wrist forcibly flexed towards the ulnar side. At times the thumb was drawn inwards till its tip appeared between the index and middle fingers.

No. 8 was exhibited to this Society by Dr. Hughlings Jackson, on November 9th, 1875, and it is by Dr. Jackson's courtesy that I have been enabled to make observations upon it. In 1866 this patient suffered from right hemiplegia, the result of a blow on the head. He completely recovered and followed the occupation of a time-keeper, writing the record of the attendances of workmen in a book. In 1873 he had a trouble with his pen, and says, "When I got hold of a pen I could not let go of it again; I thought it was cramp." In 1874 genuine spasm commenced, and by the beginning of 1876 it was so violent that he was obliged to

lie upon the arm at night to keep it quiet. The right arm measured round the biceps an inch and a half less than the left. *There was marked wasting of the extensors on the back of the forearm, which gave the degenerative reactions when tested with electricity. He was utterly unable to extend the right wrist, or fingers, or to supinate the hand, and there was diminished sensibility in the region supplied by the radial nerve.* On forcibly extending the wrist joint, some grating was audible in it. The spasm was all in the direction of flexion and pronation, and the limb was never at rest until the extreme of movement in these directions had been reached—until, in point of fact, those muscles which were the seat of spasm could contract no longer.

No. 9 was the case of a little girl, æt. 10, who had spasm of the right leg, chiefly, and to a less extent of the right arm. This condition had followed a convulsion when she was a year old. *There was diminished irritability and wasting of the tibialis anticus and peronei muscles,* and the spasm was most noticeable in the muscles of the calf and the tibialis posticus. The spasm of the hand is chiefly of the interossei. Writing was a very difficult matter, and she preferred to use the left hand, and wrote backwards. The intelligence was perfect.

No. 10 was a university student, of high intelligence, æt. 23, who had been troubled from birth with slow spasmodic movements in both hands, but especially of the left thumb. The left arm was decidedly the smaller of the two, and its muscles were all less irritable to faradism than those of the right. *There was marked wasting of the muscles of the left little finger, and impaired sensation in the region of the hand supplied by the left ulnar nerve.* Writing was a serious trouble to him, and demanded an immense mental effort. The spasm of the right side had lately extended its area, and occasionally affected some of the muscles as high as the shoulder. This extension had been induced by his increased efforts to write while preparing for his examinations. All attempts to write produced the extreme state of spasm in the right arm. There had been no extension of the spasm on the left side.

Case 11 was reported at length in the 'Practitioner' for 1873. It was a case of violent spasm of the right arm, which had commenced as ordinary writer's cramp while the patient was working inordinately hard as a writing clerk. The irritability of the muscles was not tested, since at that time I did not appreciate the importance of doing so.

It is a noteworthy fact that in four of these cases of spasm there were observed groups of muscles which were especially weak or paralysed, and in three out of the four the spasmodic movement was in that direction, which the especially weakened muscles could not oppose. In the three hemiplegic cases, in addition to the primary cause of the hemiplegia, there was a secondary paralysing lesion, either in the cord or nerve trunk, affecting a smaller group of muscles, so that we were confronted, as it were, by a paralysis within a paralysis. Now, if this secondary paralysis be not a prime factor in the causation of the spasmodic movement, it is, at least, highly probable that it determines the direction of the spasm. Although it is commonly received that such spasms are due to disturbance of the grey cerebral matter, it is as well to look also at the peripheral aspects of the question. Provided a nervous impulse issuing from the brain be distributed in a limb to equally irritable muscles, which mutually antagonise each other, it is difficult to conceive that spasm of definite form should be produced, but should the equilibrium of antagonisation be destroyed by a secondary lesion, the production of a definite spasm is easily conceivable, especially when voluntary control is lessened by a lesion of the central ganglia.

A consideration of the peripheral condition of the limb becomes more important when we remember that in some cases of localised spasm there has been no evidence of central change (as in Case 11). It is not very hard to suppose that a normal nervous impulse may produce spasm instead of orderly movement in a limb which is exceptionally circumstanced. It is theoretically possible that the action of a disordered centre on a healthy periphery, and the reaction of a disordered periphery to a healthy centre, may

be identical in their results. Although the light which has of late been thrown on the action of the nerve centres has been very great, we must not, on that account, neglect to bestow proper attention upon peripheral conditions.

With regard to localised spasm it has been generally observed that the spasm is increased, 1, by any attempt to perform delicate actions, 2, by fatigue.

If the patient persist in performing any delicate act, such as writing, the spasm may be permanently increased, as was the case with Nos. 10 and 11. In cases of localised spasm the performance of a delicate act demands a great effort for the restraining of spasmodic movement and the steadying of the necessary muscles. This has the effect of fatiguing sundry other muscles which, in their turn, take on a spasmodic action. Thus there may be added to the primary spasm a secondary spasm due mainly to fatigue, and this secondary spasm is far more amenable to treatment than the primary. This fact was evident in Cases 10 and 11.

Case 11, which commenced as pure writer's cramp brought on by fatigue, would lead one to suspect that sometimes fatigue alone is a sufficient cause for the production of extensive localised spasm.

The *third group*, consisting of nine cases, may be called the *degenerative group*, because there is good reason to suppose that in each case the impaired writing power was but the first symptom of degenerative change. Of these patients seven were males and two females. Their ages varied between forty-seven and fifty-nine, and the sole reason for seeking advice was, with each, impairment of writing power. The seven males included three clergymen, a journalist, a merchant, a teacher of languages, and a gentleman of fortune. Of the two ladies, one was a hard working philanthropist and the other was suffering from arthritis deformans. Tremor was a marked symptom in all these cases, of both hands in two, of one hand in five, while the remaining two had fibrillary tremor of some of the interossei muscles, two had diminished irritability of the inter-

ossei without tremor. Five had symptoms referable to the right leg as well as the arm. Nos. 16 and 17 presented distinct evidence of slight cerebral lesion. No. 15 was distinctly gouty. No. 12 less positively so. No. 13 had arthritis deformans, and Nos. 17, 18, and 20 have all suffered from "rheumatism" in some form.

No. 15 may be taken as a typical example of this group.

A gentleman, æt. 54, devoted to field sports of every kind, found towards the end of 1873 a little difficulty in writing, the characters becoming tremulous and illegible. He had been in the habit of occasionally writing for two or three hours at a stretch in his various account books, game books, &c., which it was his pride to keep neatly, and his inability to continue doing so was no small annoyance to him. His writing trouble continuing, he sought my advice in October, 1874. At this time his loss of writing power was his only trouble. His general health was and always had been excellent, and he was able to pursue his various amusements as ardently as ever. In his youth he had had a good many knocks and tumbles, had broken his nose and collar bone, had dislocated his left shoulder and had loosened his left sacro-iliac articulation by a fall out hunting. He had had also a mild attack of bronchitis, and an occasional deposit of uric acid in the urine, with pain, apparently gouty, in the damaged sacro-iliac joint. On the occasion of his first visit, there was, luckily, noticeable a slight fibrillary tremor of his first and second right dorsal interossei muscles, and on stripping him the trapezius and the erector spinæ on the right side were distinctly smaller than their fellows on the left. These points were all confirmed in consultation with Sir William Jenner and Dr. Wilson Fox. There was occasionally an intermission of the pulse, and the patient had a habit of now and then sighing deeply. I am informed that this patient has never been clever with his hands in matters requiring delicacy of manipulation, and that, as a card player, he was always known to be a slow and clumsy dealer. I have had ample opportunity of closely observing this case. At the present time

his muscular nutrition and his writing power which have varied from time to time are practically *in statu quo*. The condition of the muscles seems to vary with that of the urine, and when the latter is loaded with lithates and uric acid the fibrillary tremor of the interossei is generally noticeable, and the right erector spinæ and trapezius feel softer and more flaccid than at other times. There has been frequently a long interval when no fibrillary tremor was to be seen in the interossei. At first the irritability of these muscles both to faradism and galvanism was much diminished, but at present it is normal. Had this patient been first seen at a time when neither the diminished irritability nor the fibrillary tremor could have furnished a clue, the cause of his loss of writing power would have been a great puzzle. He has used the galvanic current to his wasted muscles with undoubted benefit.

With regard to No. 12 it may be well to direct especial attention to the fact that the tremor of his right arm was much increased by putting his pectoralis major or latissimus dorsi into action, as in attempting to reach the left hand breast pocket or the left hand tail pocket of his coat. The two muscles named manifested a decreased irritability to electricity and could be felt to tremble while in action. This fact of tremor being especially produced by certain acts or by putting certain muscles into action, is one which I have observed several times, and is not without its bearing upon the phenomena of writer's cramp (see Case 55).

The *fourth group*, which I propose to call the *neuritic* or *neuralgic* group, includes nineteen cases, all of which present characteristic and well-marked symptoms. Loss of writing power, or of some closely allied function, such as sewing (21, 22), painting (24), or organ playing (23), was in each case the cause of seeking advice. This group includes twelve females and five males, and the ages range between twenty-six and forty-eight. The cause in five cases was excessive work with the hand at writing or sewing (21, 23, 26, 27, 29); in eight cases there was a history of previous

strain or injury (22, 25, 30, 31, 34, 35, 38, 39); in five cases mental worry and great general fatigue had been added to local fatigue (21, 23, 24, 31, 32); in one case a difficulty in writing had existed from youth, and in one other the condition was said to be hereditary.

In eighteen of these cases the trouble was strictly limited to the right arm, while in the nineteenth both arms were affected by playing on a stiff-keyed church organ. Many other functions besides writing were impaired in every case, and, as a rule, the power of the arm for performing all acts, whether coarse or fine, was more or less impaired. Tremor was present in nine cases (especially 38); in nine cases there was tenderness of one or more of the cords of the brachial plexus, and in sixteen there was either neuralgic pain or fatigue pain after any attempt to use the arm.

In six cases there was vaso-motor disturbance in the arm. In three there was a complicating nutrition change (21 a crop of boils on the right arm, 23 herpes zoster of the right side, 25 paralysis of the right serratus magnus). Many of these patients complained of a "queer feeling" in the arm, especially at night, a constant desire to shift its position, and an impossibility of making it comfortable in bed. Five of these patients were debilitated at the time of the onset of the symptoms, two were pregnant, and three were gouty.

It is highly probable that we have to deal, in this group, with a mild form of neuritis, or at least some vaso-motor irritability, involving one or more of the nerves of the affected limb. Many of the cases very closely resemble some of those in the next group, and they are only separated from them because—

1. The symptoms involve a wider area.
2. They have been in some cases produced without excessive exercise of any function.
3. Nerve tenderness or neuralgia was a prominent symptom.

The following case may be taken as typical of this group, and it is selected because it presents many of the features of true writer's cramp.

Miss E. T—, an artist, painting on porcelain, was sent to me in May, 1874, by Mr. Christopher Heath. She was slightly debilitated at the time, but had enjoyed fairly good health. She had dislocated her left hip when seven years old, and was accustomed to walk with a crutch-headed stick in her right hand, upon which she pressed rather heavily. Had been studying painting for five years.

Four months previously the death of her father had caused her much hard work and anxiety, and for eleven nights before his death she had got no regular rest. It was at this time she first noticed that the hand became numbed and cramped, especially at night.

In March, 1874, she obtained regular employment as a porcelain painter, and worked at her art for eight and a half hours daily. She had followed this employment scarcely for a month when one day her hand became cramped and painful, "the thumb being set fast." From this time her condition got gradually worse, and she found herself obliged to grasp her brush very tightly to prevent it from shaking. On examination there was found well-marked tenderness of the musculo-spiral nerve at the external humeral point, and a condition of hyperæsthesia in the region supplied by the radial nerve, with a particularly painful spot over the carpometacarpal joint of the thumb in the depression between the extensor tendons. She said she "could not bear to have the dress even touch the back of the wrist and hand." Extension of the thumb was painful, and he said it "gets drawn back and locked, and then snaps." Forcible flexion of the thumb also caused pain, apparently by putting the extensor tendons on the stretch, the pain being referred to the point where these pass over the end of the radius. There was a difficulty in supinating the hand. The radial nerve and the extensor muscles were very sensitive to electricity, and appeared to be in a state of exalted irritability, but the pain of the application prevented a thorough examination of them. The right thumb-nail was furrowed. There was no obvious vaso-motor change. With regard to the particular implication of the extensor muscles and the radial nerve in this case,

it may be remarked that painting on porcelain necessitates a great extension of the wrist owing to the object being held nearly vertical. By the end of 1874 this patient was apparently well, when the shock and loss of rest caused by the sudden death of her brother re-induced all her old symptoms. At this time there was tremor of the arm and tenderness of all the nerve trunks. Her treatment consisted of rest, counter-irritation over the painful nerve-trunks, with bromide and iron internally. In July, 1877, she wrote, "I am glad to tell you that my arm is quite useful again, I only feel pain or numbness when I am overtired or anxious."

This is a typical case as regards cause, symptoms, and course, and I think it would be allowed that the occurrence of spasm in the extensors of the thumb, the pain and tenderness along the course of the musculo-spiral and radial nerves, the hyperæsthesia on the back of the hand, and the grooving of the right thumb-nail, all point to mild neuritis as the probable cause of the trouble.

The *fifth group* is separated by no hard and fast line from the preceding, many of the cases in each group resembling each other very closely. It is the group of true "*writer's cramp*," and includes thirty-two cases, all males, twenty-four of whom were clerks and professional scribes, who had brought on their troubles by overwork at their profession. Their ages ranged between 16 and 48, thus:

Under 20	1 case.
Between 20 and 30	8 cases.
„ 30 and 40	16 „
Over 40	7 „

The general health of these patients was good, with the exception of 44, who had a mitral murmur and an occasional cough, 47, who had a cough, 61, who had "weak nerves," and 69, who had had sunstroke in India.

It is in this class that one has had to search most closely for peripheral evidence of mischief, and the chief evidence afforded has been that of excessive or defective irritability, as tested by faradism, of some of the muscles used for

writing. This altered irritability of muscles in writer's cramp has been noticed by other observers, among whom are Zuradelli in Italy, Erb in Germany, and Gowers in this country. Evidence of muscular derangement is sometimes afforded also by the presence of fibrillary tremor after or independent of movement, even when no change of irritability is observable. In many cases more than one muscle which is indispensable for writing was found involved, and taking the whole of Class 5 we find a special derangement

Of the interossei . . .	18 times.
Of extensors of thumb . . .	10 „
Of flexor brevis pollicis . . .	7 „
Of abductor pollicis . . .	7 „
Of flexor longus pollicis . . .	4 „
Of adductor pollicis . . .	3 „
Of opponens pollicis . . .	2 „

All the muscles of the forearm were found more or less involved on two occasions.

On examining the evidence of special muscular derangement afforded by all the seventy-five cases of impaired writing power with which we are dealing, it is found that—

The interossei were especially involved . . .	32 times.
Extensors of thumb . . .	16 „
All the muscles of forearm, more or less . . .	15 „
Flexor brevis pollicis . . .	15 „
Abductor pollicis . . .	11 „
Flexor longus pollicis . . .	7 „
Adductor pollicis . . .	5 „
Muscles of little finger . . .	3 „
Deltoid . . .	2 „
Supinator brevis . . .	1 „

In every case of impaired writing power which I have seen, there has been evidence, more or less marked, of derangement of one or more of the muscles used for writing.

I have elsewhere ('Text Book of Electricity,' p. 189) attempted to analyse the act of writing, and have shown that for perfect writing a large number of muscles is employed. Of these muscles some are used for the prehension

and steadying, and others for the moving of the pen; to the former class belong especially the interossei, the abductor and opponens pollicis, the flexor brevis pollicis, the extensor primi internodii (which maintains the necessary phalangeal angle), and the supinators of the hand which serve to poise it in its necessary position (especially, probably, the supinator longus). The stroke-making muscles are mainly the flexor longus pollicis, the extensor secundi internodii, the extensor indicis, and possibly the first and second interossei also, but whether a muscle can at the same time hold a point steady and produce movement round it, is a physiological question which, as far as I know, has not yet been answered.

The muscles whose healthy action is most necessary for perfect writing are those of prehension and steadying, and it will be seen that one or other of these muscles was involved forty-two times out of a total of fifty-one instances of muscular involvement in Group 5; and if we take the whole of the seventy-five cases (excluding fifteen, in all of which all the muscles of the arm were involved) we shall find that these muscles were implicated seventy-four times out of a total of ninety-five instances of special muscular derangement.

It is not contended that all muscles except those the irritability of which was altered were healthy. The test of faradism enables us to detect, probably, the chief defaulters only, while those muscles whose perverted nutrition is not so far advanced escape detection.

It is necessary, perhaps, to say a few additional words as to the evidence of muscular derangement afforded by different cases.

1. It will be often observed that the patient has altered his method of pen prehension, and although he cannot write with his pen in one position, he can do so tolerably well by holding his pen in some manner more or less grotesque. This is one of the earliest symptoms of writer's cramp, and points to the fact that one or more of what may be called the normal muscles of pen-prehension is incapable of doing its work efficiently. Plate XI, fig. 1 (Case 19), shows the method adopted by a patient whose first right dorsal interosseus

was the seat of fibrillary tremor, and who was unable to keep his forefinger on his pen.

If the pen is grasped very tightly, as usually is the case, we may suppose that the office which should be performed by small muscles, such as the flexor brevis pollicis, and the first dorsal interosseus, has devolved upon larger muscles, such as the flexor longus pollicis, and the deep flexor of the fingers, which are less adapted for delicate accommodation.

If the forefinger rise off the pen or slip up the penholder during writing, it is by reason of the failure of the first dorsal interosseus. The "giving in" of the phalangeal angle of the thumb points to a derangement of the extensor primi internodii.

A very common early symptom of writer's cramp is an inability to keep the hand upon the paper. The hand fails to accommodate itself to the surface to be written on, and oscillates to and fro in a very characteristic manner. This phenomenon, which has long been a puzzle to me, is due, I believe, to a failure of the supinator longus to steadily maintain that degree of contraction which is necessary for keeping the hand in the semi-prone position. Directly this degree of contraction is exceeded, flexion of the elbow results, the action of the supinator longus being to commence the act of supination, and then to flex the elbow. This unsteady action of the supinator longus illustrates what I have often noticed, viz. that given any cause for unsteady action in a muscle, that unsteady action will be most manifest in conditions of semi-contraction, and least manifest when the muscle is either contracted to the full or passively stretched by the powerful action of its antagonists.

2. Evidence of muscular derangement is afforded by inquiring into the other acts besides writing which are interfered with. If a muscle be really deranged it will show its incapacity for work whenever called upon. This is very true, but, nevertheless, great caution is necessary in investigating this point. First, it is evident that there are two kinds of muscular action, *rapid action* and *prolonged action*. A muscle may exhibit *great force* or *great staying*

power. Thus we frequently see that a drunkard who has almost strength enough in his arm to fell an ox has not sufficient steadiness to shave himself. In writer's cramp it is the staying and the steadying power rather than the power of rapid and forcible action which is at fault. The conclusion that "the patient can do everything but write" is often rather hastily drawn from the fact, for example, that he can cut his dinner or play the piano without difficulty. A little consideration will show how widely these acts differ from writing. A dinner-knife is held by flexing the ring and little fingers into the palm, and the first dorsal interosseus if used at all is only called upon for a momentary contraction when the food is being cut. Again, the positions of the hand in writing and piano playing are quite different. The strain in piano playing falls upon the extensors of the wrist and fingers and the flexors of the elbows, and it is evident that the momentary contraction necessary for striking the key could be effected by a muscle which might not be able to steady a pen.

A patient will often assert that he has no trouble except with writing, because any other trouble he may have is insignificant in the annoyance which it causes him. Diligent search must be made for signs of muscular impotence by investigating those acts which necessitate a steady and prolonged contraction of the muscles or group of muscles which we suppose to be deranged. Thus, No. 60 had a difficulty in moving the regulator of his watch with the point of a penknife. No. 50 at first denied any trouble except in writing, but subsequently admitted that in holding a teaspoon he found that his forefinger slipped up the handle, and that in his business (banking) he found great difficulty in pinning together the two halves of a bank note. These two facts, his mode of failure in writing, and the test of faradism, all pointed to derangement of the first right dorsal interosseus. No. 53, who had lessened irritability in the flexor brevis and adductor pollicis, had a difficulty in holding a coin between the thumb and the forefinger, or between the thumb and the head of the fifth metacarpal bone.

If a patient be asked simply to move the interossei or other affected muscles, the ease, freedom, and thoroughness with which he does so is often in striking contrast on the two sides. There is usually no difficulty in discovering some muscular movement other than writing which is impaired.

A patient may be almost absolutely incapable of using a pen, but may be able to write tolerably well with a pencil. There are several reasons for this: (*a*) it is possible to bear some weight on the point of a pencil, which may be thus made to serve the same purpose to a sufferer from writer's cramp that a staff does to a cripple. (*b*) The method of holding pencil and pen (especially if the pencil be a short one) is often very different. (*c*) The point of a pencil never hitches in the paper. No. 69, who had great irritability of the flexor longus pollicis, found that owing to the spasmodic contraction of that muscle, the pen rotated and the edge of the nib hitched in the paper. With a pencil this rotation did not signify. (*d*) With a pencil there is not the constant fear of blotting and spoiling the manuscript that there is with a pen, and the scrivener is consequently more at his ease.

There are certain differences too between drawing and writing. In drawing the action of the pencil is more momentary, and there is not the same necessity for continued steadiness. The slope of the desk is often different for the two actions, and this causes a difference in the tension of the extensors and the supinator longus. No. 35 asserted that he could sketch without difficulty; a close inspection of the lines which he made with a pencil showed, however, that these were nearly as tremulous as those of his writing; this fact illustrates the importance of always confirming the statements of a patient.

3. The occurrence of associated or consentaneous movements, such as movements of the left fingers while the right hand is writing, may always be taken as evidence of muscular weakness. They only occur during the performance of that act which the patient finds difficult.

4. Depressed or exalted irritability is certainly a sign of

muscular derangement. It was observed in all but four of the cases in Group 5. The method of testing pursued was that recommended in my text-book of electricity, and I have no doubt as to the accuracy of the observations, since most of them were repeated and confirmed. The altered irritability of muscles in writer's cramp being an undoubted fact, it is of the greatest importance to determine what pathological changes may give rise to this altered irritability. In paralysis from cerebral lesions the irritability as a rule remains unaltered, but is, exceptionally, slightly increased or diminished. In Group 3 (the degenerative group) diminished muscular irritability was noted five times, and in each of these cases there was reason to suspect sclerosis of the cord. The diminished irritability was most marked, however, in muscles which had been overworked. In locomotor ataxy I have seen the irritability of muscles both diminished and increased. Dr. Gowers has noticed an increase of irritability in chorea. In this disease many muscles are presumably overworked. In Case 73, where impaired writing power was the first symptom of general paralysis, certain of the muscles of the thumb showed great exaltation of irritability, but the muscles had certainly been overworked. In Case 60 was found an increased irritability of the first dorsal interosseus, apparently dependent on a swollen condition of the ulnar nerve. Lastly, diminution of the irritability may be quickly and certainly produced by a prolonged faradisation of a muscle. This is an experiment which any one may try upon himself. Thus we see that alterations of irritability accompany many conditions both central and peripheral. There is no necessity for assuming that central change exists in every case of altered irritability, and we have no warranty for such an assumption in the absence of independent evidence of central change. It is certain that alterations (especially depressions) of irritability occur in muscles which have been overworked, and notably in those which have been subjected to prolonged strain. This condition occurring in overworked muscles I have called "fatigue." The term seems to me a convenient one because fatigue is familiar to us all,

Page 128.

action ting.	Tremor.	Neuralgic pain.	Fatigue pain	Treatment.	
pronate ly with-	Fibrillary tremor of 1st right dorsal interosseous	—	After writing of ple u-	Belladonna liniment; bromide internally.	
poon his s up "the ot pin a her with- Dislikes -stick	Very slight at times	—	" and ng, ing all. rs. ith are his the	Rest; friction; blisters to painful nerves; injections of atropine (gr. $\frac{1}{100}$ hypodermi- cally daily). To take horse exercise, and use iodide and bromide of potassium. At the end of 3 months the writing was no better. November, 1877.—Much improved. His memory has quite recovered.	
ny move-	After writing or other exercise	—	—	Strychnine; galvanism; rest. He writes in July, 1876:—"I am really a great deal better."	
irritable radism	—	The ball of thumb after writing	Very marked with after writing le- me. is a	The galvanic current and rhythmical exer- cise, which patient carried out for himself with very great judgment and persever- ance. The feeling of fatigue was quite re- moved, and the ease of writing very much increased. The objective features of the writing are very slight.	
between ld of 2nd pal bones of great	—	—	After writing g. line ano	Bromide; friction; injection of atropine; galvanism. Blister over median nerve. Improved very rapidly.	
	—	—	ith a Optic		
	—	—	Marked after writing	Got quite well with rest.	
any move- especially drawing supinating	Marked after writing or exertion	—	After exerci- right up to the shoulder	Tonics; liniments. When first taken ill he kept the arm in a sling for 3 weeks, but found the arm much weaker afterwards.	
is arm out s weary." t his baby d tremble	Marked after exercise	—	Marked	Has a so by merus, of his slope and not hand	Is scarred from neck to wrist by using a chain-band. Tonics, galvanism, rhythm- cal exercise improved the general sense of weariness and the tremor. The fatty tumour was removed on August 5th, but its effect on the writing is not known. Hypodermic injection of gr. $\frac{1}{100}$ of atropine was without result.
hammer with manipulate e knots	None. Has the "fulgurant" pains ataxy very marked in both arms	—	upper n with n only n then ired in	The galvanic current relieved his pain very considerably, but did no further good.	
se his right t hold his easily as	After writing	—	Marked	potent. Bromide; rest; arsenic; and friction; bli- sters to spine. The symptoms of general paralysis ultimately declared themselves.	
	—	—	—	with the e right. t bowls t hand. and in The left and the ver, and	Not requiring treatment. He was kindly sent to me by Dr. Reynolds as a curiosity, that I might examine him.

but whether it be primarily dependent upon change in the muscular fibre, the nerve end, the nerve trunk, the cord or the brain, there is not sufficient evidence to show. It seems to me reasonable to suppose that while in some cases fatigue may be the sole cause of the trouble, it probably is more often merely a determining cause of its localisation.

The result of such alterations in irritability would be to render the affected muscles uncertain in their response to the stimulus of the will.

The occurrence of altered irritability in muscles which have been especially overworked is strikingly illustrated in the following case (55), which I am very glad to be able to quote, because Dr. Reynolds was a witness to the facts.

Mr. T. H. B—, a government draughtsman, æt. 27, suffering from writer's cramp, was examined by me in consultation with Dr. Reynolds, on May 18th, 1877. He had been working very hard at his profession. On testing his muscles we found a very marked excess of irritability in his *right* abductor and opponens pollicis; and a decreased irritability equally marked and decided of his *left* first dorsal interosseus. As there was something evidently anomalous in the case I asked the patient to see me again when I could give unlimited time to his examination. The facts previously observed were confirmed at the second examination, and on questioning him as to the cause of his trouble I found that it was not due to writing but to excessive work at making plans, at which he had laboured for as much as thirteen hours a day. He had used a protractor and scale with his left hand, and an instrument called a "pricker" with his right. This would entail a constantly repeated contraction of his right opponens and abductor pollicis, and a prolonged strain of his left first dorsal interosseus (see Plate XI, fig. 2). Thus, what at first seemed rather to militate against the theories I had advanced, proved to be a striking confirmation of them. This patient had slight tremor of both hands, but especially of the right, when the abductor pollicis was put in action.

I am unable to state with any weight why it is that in some cases irritability is increased and in others diminished. I am inclined to the opinion that increased irritability shows an early, and decreased irritability a late stage of the same condition.

5. Indirect evidence of muscular derangement may be afforded by derangement of the sensory branches of mixed nerves which supply the muscles implicated. Neuralgia as well as numbness of the region of the hand supplied by the radial nerve was noted several times, and in all these cases the extensors or supinators were implicated. It is not a little remarkable that I have seen no case in which the sensory branches of the ulnar nerve have been involved, notwithstanding that the muscles of the hand supplied by this nerve are implicated more frequently than any others. An explanation of this may possibly be found in the fact that the twigs of the deep palmar branch of the ulnar nerve which supply the muscles affected are completely separated from the sensory branches of that nerve, so that any molecular change brought about by fatigue in the former branches would be well removed from the latter.

The radial nerve, on the other hand, is so largely concerned in the supply of the ligaments, joints, and tendons of the overworked part that the frequency of its implication is no matter for astonishment.

It has been very often noticed that the sense of muscular contraction is far less acute in the implicated muscles than in their healthy fellows, although the fact has not been systematically recorded. Patients have almost always said that the sensation caused by the application of faradism "is quite different on the two sides." Some of the phenomena of writers cramp have been referred to impairment of the muscular sense or to muscular paræsthesiæ, and this observation gives colour to such a supposition.

Nerve tenderness, it will be observed, was one of the commonest phenomena of the whole series of cases.

The *sixth* and last group of cases of impaired writing

power, which may be called the *anomalous group*, consists of four cases only, bearing no resemblance to each other.

The first (No. 73) was a case of locomotor ataxy, showing itself in the arms before the legs. The patient was a tailor's packer, and worked mainly with his arms. It is an example of the power of fatigue to determine the localisation of symptoms.

The second (No. 74) was a case of general paralysis of the insane, in which impairment of writing power was the first symptom. The patient was a secretary, and has since manifested all the symptoms both mental and muscular of general paralysis. This is another instance of the power of fatigue to determine the localisation of symptoms. There was in this case nerve tenderness and tenderness of the cervical region of the spinal column.

The third case (No. 75) was that of a clerk, who was strangely left handed. He wrote, held a violin bow, and manipulated the stops of a flute with his left hand, he used a knife and a hammer with his right hand, and was a "right-handed bat" at cricket. It would seem as if he preferred to use the left hand for delicate manipulations, and the right for coarse acts; no cause could be detected for this. The left hand and leg were both rather bigger than the right, and the left boot was the first to wear out; there was no transposition of viscera. The muscles of the right arm and hand were somewhat less irritable to faradism than those of the left.

No. 72 is placed in this group because it was uncertain how much of his trouble was due to a fatty tumour over the deltoid muscle.

Reviewing these seventy-five cases collectively there are a few points to which attention may be directed.

What evidence of the exact condition of the patient is to be obtained by examining the handwriting? Very frequently the handwriting is perfectly normal, and an inspection of it would never lead one to suspect that its production had caused the scrivener much labour and trouble.

In the paralytic group the change in the writing was only slight, except in Case 3, where there was anæsthesia as well as paralysis. In each case the writing was slowly produced.

In the senile group the writing was characteristic, and presented a general uniform tremor.

In the neuralgic group the writing was more uncertain than tremulous. In the writing of Case 37 the down strokes bore away remarkably to the left. This was due to the forcible contraction of the pronators unopposed by the weak supinators.

In the group of true writer's cramp the writing was very varied in character. Inequality in the thickness of the strokes shows unequal pressure with the pen, and here and there one may see how the nibs of the pen have been forced asunder. The writing of No. 61 showed a special uncertainty in the up-strokes due to a failure in the free extension of the forefinger. No. 72 had a difficulty in keeping his writing on the line; this was apparently due to a want of free action of the deltoid and triceps. The movement of the hand from left to right was effected in this case by making a pivot of the styloid process of the ulna and adducting the humerus, so that the lines instead of being straight were all segments of circles.

Implication of joints was noticed in no less than 21 cases (See 1, 2, 4, 8, 13, 15, 17, 18, 20, 22, 23, 24, 25, 35, 43, 45, 46, 52, 58, 66, 71), the affection was generally called rheumatic; sometimes it was a consequence, no doubt, of the strained position in which the joints had been retained for prolonged periods; in other cases it was probably a joint affection of neuropathic origin. The snapping or loud spontaneous movement of one of the joints of the thumb was observed five times (24, 25, 46, 52, 58). A feeling of "weakness" in front of the elbow was noted five times, and in one case it was accompanied by a snapping sound.

Another point of great interest was the frequency with which it was observed, that the difficulty in writing was inherited or had shown itself in more than one member of a family, or had become manifest very early in life. This was

observed ten times (9, 10, 15, 19, 30, 44, 45, 68, 70, 75). The case of congenital left handedness is of great interest, because the peculiarity was most manifest in the performance of delicate acts. Want of freedom in such an act as writing, may be due to the want of proper development of the small intrinsic muscles of the hand. These muscles may very well show occasionally a defective development just as the muscles of the external ear are sometimes excessively developed.

The most interesting case of heredity or family tendency is No. 70. G. W—, a young doctor of medicine, who had been working exceedingly hard suffered from a true attack of writer's cramp, the result of combined general and local fatigue. He was also short sighted, and any attempts to read without spectacles produced at the time of his trouble symptoms of headache and vertigo, which were also produced by attempts to write. Any attempt even to write his signature ended in failure, and produced fatigue, pains up the arms, and a sense of distress and vertigo, although he could dictate to another without difficulty of any kind. When I saw this gentleman he could write perfectly well, but I tested his muscles as a matter of curiosity. There was doubtful excess of irritability of his *first and second right dorsal interosseous muscles*, but diminished irritability of the *abductor and flexor brevis minimi digiti*. This he accounted for by the fact that he had been sketching for a long time on the previous evening, holding his drawing block in his left hand, and steadying his right hand by prolonged abduction and flexion of his little finger. There was tenderness of the right ulnar nerve. Short sightedness is common in his family, and one of his sisters has had vertigo and fatigue symptoms from reading without spectacles.

This case forms a very interesting link between writers' cramp and a case reported by Mr. R. B. Carter in the eighth volume of the Clinical Society's 'Transactions.' Mr. Carter's patient had been reading hard at the university. He was troubled by attacks of vertigo which supervened whenever he attempted to read. His case was regarded as

one of cerebral disease by more than one physician, but he was immediately and completely cured by a pair of concave glasses which relieved the internal recti of the strain imposed upon them in reading. This case must impress us with the fact that central symptoms may be merely reflex, and mainly dependent on peripheral causes.

Definite evidence of change in the nerve centres was observed only a few times. In three out of the five spasmodic cases there had been a previous hemiplegia, and in all the degenerative cases there was reason to suspect a chronic change in the cord, or brain, or both. The same may be said of two of the three anomalous cases. Among all the other cases there were only three in which there were unmistakable signs of involvement of the brain or cord. These were No. 70 given above; 68 which was a similar case, and 69 who had had symptoms of sunstroke. All three had altered irritability of muscles, distinctly evident.

Tremor was a symptom which was very frequently observed, but this I think, need not, necessarily be regarded as certain evidence of involvement of the spinal cord in the absence of other symptoms referable to that centre. Tremor is certainly very often caused by lesions of the nerves themselves. Tremor of a limb is generally most marked when an attempt is made to use paralysed or paretic muscles.

The after history of cases of writer's cramp would be likely to throw much light on the pathology of the disease. I have been unable, however, to acquire any facts in this direction.

Writer's cramp has been spoken of as a disease of faulty co-ordination, and most writers on the subject have referred the symptoms to a lesion of the centres which control the co-ordination of the muscles used in writing.

In Ziemssen's 'Cyclopædia of Medicine' writer's cramp is included in the volume devoted to diseases of the peripheral cerebro-spinal nerves, but Erb, who writes upon the subject, says, "In the present state of our knowledge we are justified in placing the seat of the cause of the typical forms

of writer's cramp in the central nervous system, although we are not in a position to locate it with precision."

In Reynolds' 'System of Medicine' writer's cramp is classed with the general nervous diseases, and the disease itself is regarded as a failure of co-ordination merely.

There can be no doubt that a want of co-ordination is the proximate cause of the symptoms of writer's cramp, for it is evident that the muscles fail to work orderly together. Are we, however, justified in assuming the existence of a special co-ordinating centre for the controlling of the act of writing; and is there any evidence to show that this centre (supposing it to exist) ever gives way leaving the periphera in a state of perfect health? I think not, and mainly for the following reasons.

1. Because I have never seen a case without evidence of peripheral change, and in the great majority of cases there was no reliable evidence of any pathological change except at the periphera. The evidence of peripheral change consisted of—

- A. Definite muscular paresis.
- B. Definite muscular spasm.
- C. Localised tremor.
- D. Fibrillary tremor of certain muscles.
- E. Alteration of muscular irritability.
- F. Localised pain.
- G. Nerve tenderness.

One or more of these conditions was always present. The writer's cramp of the text books, in which failure of writing power is the sole symptom, I have never seen.

2. If there be a co-ordinating centre for writing, then the conclusion seems irresistible that co-ordinating centres can be created as it were by education. Writing is no more an essential attribute of man than any other muscular faculty which he may acquire by education. No two people probably, co-ordinate the same muscles in precisely the same way for the purpose of writing, and it is well known that it is scarcely more difficult to write with the toes than with the fingers. The co-ordination of writing, which we acquire only

by years of painful practice, is clearly to be distinguished from those co-ordinations such as the symmetrical movement of the two eyes, which are wholly independent of education, and which we are almost powerless to disarrange.

3. The fact that writer's cramp is always of more or less gradual growth, and is never suddenly established, militates very strongly against the idea of a controlling centre, which, whether congenital or acquired by education, would surely be liable to the occasional accident of sudden extinction, just as the centre for language is sometimes suddenly extinguished. The impulses which move our writing muscles proceed, probably, from different points in the cortex cerebri, but there is no evidence to show that these impulses are, in their transit to the muscles, welded as it were into harmony by converging on any "centre."

4. The fact that the left hand (if used for writing by a sufferer from writer's cramp) is liable to be affected in the same way as the right hand, has been used, especially by Duchenne, as an argument in favour of the disease being due to a central lesion. I am glad to find that Dr. Gowers in a recent paper¹ considers that the liability of the left hand to suffer has been overestimated for I am decidedly of the same opinion. It must be remembered, however, that a patient who has disabled his right hand by excessive writing, has often by the great mental efforts which he has used to goad his unwilling muscles, as well as by the mental worry produced by his condition, been reduced to a state of general nervous exhaustion, which would certainly make him very vulnerable to the effects of local fatigue. That writer's cramp should have occurred at all is certainly evidence of vulnerability to fatigue, and it is to me no matter of surprise that the muscles of the left hand should succumb to the excessive mental stimulus applied to them. We must bear in mind, too, those associated movements which are so commonly observed in writer's cramp, and which may be only a form of reflex action. If, as in some cases of writer's cramp, the patient

¹ 'Med. Times and Gaz.,' Nov. 17, 1877.

is unable to write without moving his left hand in sympathy with his right, it is impossible not to admit that the left hand has been fatigued to some extent by these movements, and insidiously prepared, as it were, for the fully developed disease.

We have in the occasional spreading of writer's cramp from one hand to its fellow, a phenomenon analogous to the sympathetic inflammation of one eye after an injury to the other, and to the so-called *neuritis per saltum*, as described by Nothnagel, in which after inflammation of a nerve on one side of the body, the corresponding nerve may be attacked without the implication of the spinal cord.

It is not more difficult to believe that something of this kind occurs in "writers cramp," than that a central lesion is transferred from one hemisphere of the brain to the other or from one side of the spinal cord to the other, without the implication of the intermediate tract of nerve tissue.

5. Whether there be, or be not, a controlling centre for the act of writing. It is perfectly clear that the co-ordination of writing or of any educated movement may be upset by a peripheral lesion causing an uncertainty in the response of the muscles to the mental stimulus. This want of co-ordination only occurs when muscles are paretic or excessively irritable, and not when they are completely paralysed, for the action of a muscle which is paralysed soon ceases to enter into the mental calculation.

Case 60 may be quoted as an example of writer's cramp arising entirely from a peripheral lesion.

Mr. H—, a manufacturer, æt. 43, a man of great energy, consulted me on April 8th, 1877, for a difficulty in writing, from which he had suffered for two or three years. He had never done much writing beyond a certain amount of book-keeping. His interossei moved in a very jerking, uncertain manner. He could not write, nor lift a tea cup between his thumb and forefinger, and had signally failed when trying to regulate his watch with the point of his penknife. His general health was perfect. On testing his muscles the first right dorsal interosseus was found remarkably irri-

table, responding to a current which had no effect upon the corresponding muscle. On examining his hand and wrist a distinct swelling was found under the flexor carpi ulnaris directly over the course of the ulnar nerve, and which appeared to be a swelling of the nerve itself. With regard to this a history was elicited that he had found considerable trouble with his sleeve studs which were very large, the ornamental boss being as big as a florin and the under part as big as a sixpence. This under part exactly impinged upon the ulnar nerve; he had often found his studs in the way when writing and had reproved his shirt maker for putting the stud holes in such an inconvenient position. It seemed difficult not to connect the stud with the swelling on the nerve, and this with his difficulty in writing and the great irritability of his interosseous muscle. This gentleman had been advised to abandon his business, and had worn a seton in the back of his neck. He had rubbed in belladonna until his pupils had become dilated and had tried every form of galvanism, as well as Turkish baths and Swedish gymnastics. He had taken ergot, bromide, and strychnine. I advised him to abandon his sleeve studs, to leave off writing for a time, since other muscles showed signs of irritability, and to apply blisters to the seat of the swelling. When last I saw him (February, 1878) he was somewhat better. The swelling over the ulnar nerve had disappeared; the exalted irritability of his interosseus muscle, though still present, was less marked; and his hand writing had undergone some slight improvement.

In previous writings on this subject I have spoken of writers cramp as a "fatigue disease," and as due to a condition of irritable weakness in certain muscles, generally brought on by overwork, but occasionally by other causes also. The word muscle must be understood to be a clinical expression, for the muscle with its motor nerve. I am inclined to adhere to the word "fatigue" as a convenient expression for an easily recognisable and familiar condition of whose pathology we are uncertain. I am inclined to think, however, that occasionally "fatigue" is the expres-

sion of hyperæmia or mild inflammation of a motor nerve, and that a similar condition of "muscle" may be produced by overwork or by accidental causes, such as cold, strain, "rheumatism," or injury. In this way we reconcile the two classes of writers cramp, that in which there is a history of overwork, and that in which there is not.

The law of fatigue, as enunciated by Professor Haughton, is "when the same muscle or group of muscles is kept in constant action until fatigue sets in, the total work done multiplied by the rate of work done is constant."

Now we have seen that fatigue especially attacks those muscles which are subjected to prolonged strain (in which the rate of work done is at a maximum), and I believe that the relative frequency of "writers cramp" as compared with other professional ailments is due to the fact that a prolonged strain of certain muscles (those of prehension and steadying) is inseparable from the act of writing.

If I were asked to assign to writers cramp its proper position in the catalogue of diseases I should couple it with neuralgia; that is with a disease, all the phenomena of which are local, but the local phenomena of which we recognise as being due, not only to conditions affecting the sensory area involved, but also to molecular change affecting any part of the sensory fibre, whether before or after its junction with its nerve centre.

With regard to the *treatment* of impaired writing power, it must be borne in mind that we are dealing only with a symptom of disease, and that, if we wish to alleviate the troubles of the patient, an accurate diagnosis of the cause is first of all necessary. I have known a patient with a simple paralysis of the hand warned on no account to touch a pen, a piece of advice which did him probably no good, and which caused him a large amount of inconvenience and mental worry. I have given the details of another case in which the patient wore a seton in his neck to cure a lesion situated an inch above his wrist joint. These examples are enough to show that a careful study of each individual case, with a view to exact diagnosis, is very necessary.

In paralytic cases the indications for treatment are three-fold :

1. To favour the healing of the lesion.
2. To preserve the nutrition of the paralytic muscles.
3. To force, as it were, the passage of the lesion for the mental stimulus.

The first indication, if the case do not come within the province of the surgeon, may be met by the exhibition of tonics, or perhaps of iodide of potassium, and possibly by the employment of counter-irritation.

For the fulfilment of the second object we must, above all things, promote nutrition by warmth, friction, baths, douches, and passive movement of the muscles, and as a further help, we may call in the aid of electricity, employing that current to which the muscles most readily respond, which in most cases is the galvanic.

To effect the third object of treatment we must incite the patient to make a moderate use of voluntary effort, and tell him to try his best to move the paralysed parts. We may also do something by passing a galvanic current through the nerve from a point above the lesion to the paralysed muscles.

If the impaired writing power be caused by definite and widespread spasm, we must first ascertain whether any muscle or group of muscles is the seat of paresis or paralysis, and is thus indirectly causing the spasm by its failure to antagonise. These paralysed muscles must receive the treatment recommended for the previous group of cases. If there be contracture of any of the muscles, then steady extension by means of a suitable apparatus may be tried (as in Case 7). Any work which involves a great effort to steady the limb must be absolutely prohibited, otherwise the spasm will undergo a steady growth and extension. Sleep must be encouraged by the exhibition of bromides; and the excessive use of nervine stimulants must be forbidden. Lastly, the galvanic current combined with a rhythmical exercise of the affected muscles is to be employed. This method of treatment I have found of some service, Nos. 7 and 8 were

slightly benefited (there was reason to believe that No. 8 did not always take the trouble to exercise a maximum amount of control over his limb); No. 9, although congenital in its origin, was benefited most markedly, and the patient having purchased a battery for himself, found that when he was accidentally prevented from using it, his symptoms returned; No. 11 was most markedly benefited.¹ I have employed hypodermic injections of morphia and atropine, but without any obvious results.

In the treatment of the degenerative group it is, of course, important to recognise the fact that the loss of writing power is probably only the first symptom of a degenerative change, which may eventually be more or less widely spread. It is needless to put any very rigid restrictions on the patient as to writing. He should be told to look upon his trouble as a warning to take life more easily than, probably, he has hitherto done. Careful search is to be made for evidence of gout, or for renal, vascular, or cardiac degeneration.

The treatment of the neuralgic or neuritic group must be directed to the constitutional condition as much as to the local trouble. We must see that the patient is removed from causes of physical overwork or mental worry. Absolute rest for the damaged limb is necessary, but in prescribing this we have to guard, especially with women, against engendering, by enforced idleness, a morbid habit of mind. We must search for and treat anæmia, gout, rheumatism, and sleeplessness, and see that the patient's surroundings are all conducive to health. Much can be done by local treatment such as friction, local vapour baths, and stimulating applications. We must be careful that friction, especially if carried out by professional shampooers, is neither too severe nor too prolonged; if the nerve trunks be tender they should be blistered or painted with iodine, a mode of treatment which rarely fails to give relief. A course of warm bathing at Bath, or still better, at some elevated health-resort, such as St. Moritz, in the Engadine, is likely to be

¹ See 'Practitioner,' vols. x and xi, 1873.

of great service. I have never seen any decided benefit from the application of electricity in these cases, the patients are rarely able to tolerate its application. The course of these cases is very chronic, but if the patient be young, I believe recovery to be the rule.

Lastly, as to the treatment of that group which includes the cases we have, mainly by a process of exclusion, called *true writer's cramp*, The first indication is to obtain rest, not only for the damaged member, but also for the mind and body of its owner. No treatment is of much service if the patient cannot get rest, and the man whose hand has once broken down from overwork must be warned of the necessity of providing at all costs against overwork in the future.

Great good is often done by explaining the cause and nature of his complaint to the patient and I have more than once succeeded in relieving a sufferer of a load of mental anxiety by demonstrating to him that some of his hand-muscles were, so to say, out of gear, and that his malady was in great part at least, merely local. A right hand that refuses to obey the will is to a man of energy, and these patients are mostly men of energy, an annoyance which can scarcely be exaggerated. One of my patients was meditating suicide. Another had in a moment of passion stabbed himself in the right wrist, and others had conceived the idea that the faltering hand was the prelude of insanity.

The general health must, of course, receive careful attention, for in many of these patients it was observed that any failure of health, even a passing cold, "flew to the writing." Mental worry and sleeplessness may be relieved by bromide.

If the trouble has been definitely brought on by overwork a rest from the special work is generally sufficient in the early stages of the disease to insure a rapid recovery. A medical friend of mine had symptoms of writers cramp from writing short hand. He has given up short hand and has had no more trouble. Another friend, a journalist, has found it advisable to abandon the use of steel pens. A third, an architect, got definite symptoms of writer's cramp

from working excessively, using a hard pencil with great pressure, at the details of an architectural drawing. He left his work and went to Switzerland for six weeks, where he worked very hard at mountaineering. On his return he was no better, but six weeks later, when presumably he had recovered from the excessive fatigues of his Swiss tour, his hand got quite well and has never troubled him since, for like a sensible man he is careful not to overtax it.

There is, I think, no harm in permitting a moderate use of the left hand to relieve the right.

If the patient neglect the early symptoms of his trouble, recovery is not so easy, and it may be necessary to adopt some measures to aid nature. One of the most useful aids is, I have no doubt, the rhythmical exercise of the affected muscles. The muscles have become fatigued because the periods of their contraction have been out of all proportion to the periods of relaxation. Properly regulated exercise of function seems to be able to restore the tone of these muscles better than absolute rest, and the patient should be shown how to exercise them slowly, rhythmically, and methodically. This he should do once or twice daily, and should be warned not to fatigue himself with his treatment. Friction of the muscles with a slightly stimulating liniment is of decided service in restoring the irritability of the damaged muscles. I have seen the greatest good result from combining the galvanic current with rhythmical exercise in the manner detailed in my text-book of electricity. My belief is that the application of the current causes an afflux of blood to the muscle (the skin over it is certainly reddened), and that the well-regulated exercise of the muscle while it is in this condition has the effect of improving its nutrition. The application generally relieves the sense of fatigue effectually and at once. I have only had an opportunity of knowing the effects of this treatment in a few cases, but I may point to Nos. 51, 52, 57, 58, 59, 64, 65, as cases in which the benefit of this mode of treatment was so marked as to leave no reasonable grounds for doubting its efficacy.

I have used injections of atropine and morphia occasionally, but without any decided results. If any nerve trunks be tender a blister should be applied over them. Of the utility of this method of treatment I have no doubt.

Since the chief cause of writer's cramp is, I believe, the prolonged strain to which certain muscles are subjected, I scarcely ever recommend any mechanical contrivance for holding the pen. It is impossible to steady a pen for writing without throwing certain muscles into prolonged action, and the most that a mechanical contrivance can do is to throw the strain from one set of muscles on to another. By holding his pen in different positions the patient manages for a time to shift the strain of writing, but he generally finds that each fresh group of muscles soon gives out. Whether he hold his pen like a baton, or interlace it among his fingers, or have it strapped to one finger or to the arm the strain must come somewhere, and he finds that the flexors of the fingers, the extensors of the wrist and fingers, and even the biceps, triceps, or deltoid, soon give out as the first dorsal interosseus or the extensors of the thumb have given out before them. The patient should be warned that the persistent use of mechanical contrivances is a delusion and a snare, and that by employing them he is following that course which will end in the extension of his trouble.

If such contrivances be used only for the execution of small and occasional pieces of writing they may then be employed. We have alluded to one patient who could write only when his wrist was bound to the desk by a restraining band. Others can write better on sloping than on flat surfaces, or *vice versa*. A penholder of large diameter, soft quill or vulcanite pens, or pens with recurved nibs may give some increased facility. I have recommended a wooden ball as big as a billiard ball for a penholder, and if this ball be perforated to receive pegs in various positions, an almost endless variety of methods of pen prehension may be obtained. If the chief trouble be an obstinate rotation inwards of the pen by the constant

flexion of the thumb, then a penholder furnished with a freely movable rotating ring so that this ring may rotate instead of the pen itself will be found useful. It is always necessary to study the case carefully, and to make out accurately the cause of failure, before recommending this or that mechanical contrivance.

There is one mechanical contrivance for writing which is not open to the objections mentioned above, inasmuch as it entails no prolonged strain upon any of the muscles. This is the American type-writer, a machine which is worked by keys like a piano. This machine may be safely recommended to those who can afford to buy it; its only drawback is its noisy action.

Of all cases of writer's cramp those which are the most difficult to treat are the hereditary cases, or those which have arisen from some cause, such as cold or strain, other than overwork. My experience has been that such cases prove very unyielding to therapeutic measures.

In conclusion, I have to thank many of my colleagues and others for giving me an opportunity of examining several of the cases which are the subject of this paper. My thanks are especially due to Dr. Russell Reynolds.

DESCRIPTION OF PLATE XI.

Cases of Writer's Cramp. (Dr. Poore.)

FIG. 1. Method of pen prehension. Case 19. Fibrillary tremor of first right dorsal interosseous. (See p. 124.)

FIG. 2. Use of protracting scale and "pricker." (See p. 129.)

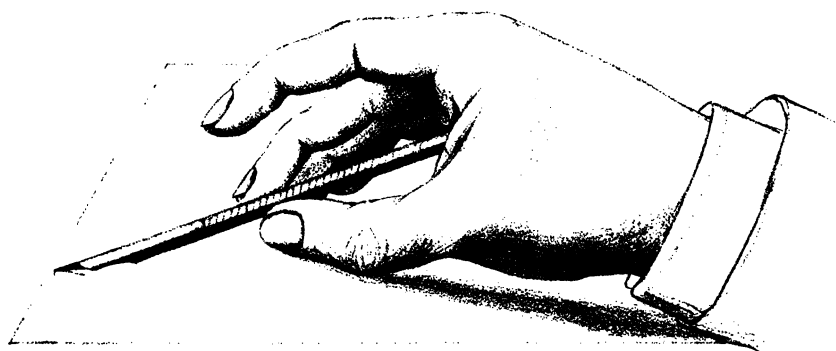


Fig. 1.



Fig. 2.

ON A CASE OF AMNESIA,

WITH

POST-MORTEM EXAMINATION.

BY

WILLIAM HENRY BROADBENT, M.D.,

PHYSICIAN TO, AND JOINT LECTURER ON MEDICINE AT, ST. MARY'S HOSPITAL;
PHYSICIAN TO THE LONDON FEVER HOSPITAL.

(Received December 20th, 1877—Read February 26th, 1878.)

IN Vol. LV of the Society's 'Transactions' for the year 1872 there is a paper by the writer on the "Mechanism of Speech and Thought," some of the hypothetical conclusions of which it will be necessary to restate very briefly, in order that the bearing of the case, which forms the subject of this communication on the question of the cell and fibre apparatus concerned in intellectual operations, may be understood.

Dr. Bastian's hypothesis was accepted which assumes that there will be a certain district or area of the surface of the cerebral hemisphere in relation with each sense, in which the sensory impression undergoes the intellectual elaboration by which it is translated, so to speak, into a perception or recognition of the external cause of the sensation. These are called "perceptive centres." At that time nothing was known of their situation, but investigations into the distribu-

tion of the fibres passing from the crus and central ganglia in the "internal capsule" and "corona radiata" to the convolutions, had led to the inference that these "perceptive centres" would be found in certain convolutions in which the radiating fibres terminated. Ferrier and Hitzig have since identified these "perceptive centres," and the former, experimenting on a monkey, localises the perceptive visual centre in the angular gyrus, the auditory centre in the infra-marginal gyrus of the temporo-sphenoidal lobe, smell and taste in or near the uncinate lobule, touch in the uncinate gyrus. These are all in the marginal convolutions, which anatomical research had indicated as receiving radiating fibres, and as being thus in direct relation with the outer world, either receptively through the senses or through the muscles. These particular convolutions, moreover, all receive their fibres from that part of the thalamus which may be called extra-ventricular, namely, the process which bends round the posterior edge of the internal capsule. It having been found that certain other convolutions were not directly connected with the central ganglia or crus, but only with other convolutions by arcuate fibres, the speculation was pursued further. It appeared probable that the association together of the different perceptions of a given external object, which would by their combination constitute the complete idea of the object, would be represented structurally by the convergence of arcuate fibres from each of the perceptive centres to some of these "superadded" convolutions. Since, moreover, the fully formed idea implies the attaching of a "name," the convolution in which the perceptions were combined into an idea would also be the *naming* centre, and would be concerned with *words* as auditory impressions and intellectual symbols.

It was not attempted to follow the mechanism of thought further than this rudimentary step, but the study of words was taken up from the opposite aspect as motor processes. With this, however, the present communication is not concerned.

On the above view of the structural arrangements for the

formation of an idea, it was pointed out as a possible occurrence that the fibres of communication from one or other of the "perceptive centres" to the "naming centre" might be destroyed by disease. Supposing this to occur in the case of the visual perceptive centre, the sight of an object would then no longer recall the name. This condition was actually illustrated by a remarkable case.

A very intelligent man, who talked fluently and well on any subject, and who wrote correctly, spontaneously or from dictation, had completely lost the power of naming anything shown to him. He could not after prolonged and painful effort name so familiar an object as a hand, or coat, or table, or recognise a letter of the alphabet, or read a word even of what he had himself written a moment before. The lesion giving rise to this condition was situated in the white substance of the left hemisphere, between the posterior extremity of the fissure of Sylvius and the lateral ventricle, where this gives off the descending cornu.

With this illustration in mind let it be supposed that there had occurred a similar rupture of the communication between the auditory perceptive centre and the *name* centre, or *idea* centre, as it may indifferently be called. It will be seen that the effects will be of a far more complex character. If a spoken word conveyed no more idea to the mind than the written word in the case alluded to, the subject of the lesion would understand absolutely nothing that was said to him; a more serious and perplexing difficulty, however, would be that he would not know what he himself was saying, his own speech would be as unintelligible to him as his own writing was to the patient whose case has been quoted. This condition was supposed to be illustrated in Case 9 of the former communication, but the patient was seen only once in consultation, and no post-mortem examination was permitted.

Another condition may be imagined, that is, lesion in name or idea centre itself, and this is what is believed to be illustrated by the case now to be related.

James S—, æt. 60, an omnibus driver, was admitted in

St. Mary's Hospital on Oct. 16th, 1877. He had been a fairly healthy man, and was described as having been particularly intelligent, a good talker, reading much, and able to write well. He had been addicted to drink in the form of heavy bouts from time to time with sober intervals. In May, 1877, he lost his wife, and had been drinking hard, with little intermission, ever since.

A fortnight before his admission he had some sort of fit, of which no reliable description could be obtained, and from that time had been unable to speak intelligibly, and had kept his bed.

On admission he was well nourished, had a good colour, a dark complexion, and curly dark hair turning grey. The expression of his face was rather anxious and inquiring.

He walked into the hospital and up the stairs to the ward guided and assisted by friends, and had free use of both upper extremities. There was thus no hemiplegia, but slight paresis of the right side of the face was observed. Later, it was discovered by the clinical clerk, Mr. Handfield Jones, from whose careful and able notes the account of the case is abstracted, that there was decided impairment of sensibility in the right half of the body.

The most striking feature in the case was that the speech was for the most part an inarticulate jargon. When questions were asked he replied, but as a rule nothing at all resembling a word could be detected in what he said. The voice was inflected naturally, and he appeared to think that he was understood. Besides answering questions he would go on talking, addressing himself to one or another of those present, the whole being an incomprehensible ramble, but at times a distinct word or phrase would slip out. He was emotional and often cried, and it was not difficult to make out that he was begging for something—probably drink; the word rum was distinguished once or twice, and he would become excited as he went on and end by crying. When excited the phrase "If you please" was several times heard distinctly.

It was difficult to make out how far he understood what

was said to him, his answers being unintelligible. He turned over in bed when told, but probably he was assisted by signs and by assistance offered him. When questioned about his symptoms he did not appear to comprehend at all what was asked. He was, moreover, obviously quite unconscious of the fact that his own speech was gibberish, and appeared to have some idea in his mind and to think that he was giving expression to it.

The pulse rate was 140, but many beats failed to reach the wrist. There was no cardiac impulse anywhere. The heart sounds were feeble; and near the apex the first was short and sharp, and either it was reduplicated or there was a slight presystolic murmur; the second sound was inaudible at the apex. The breathing was hurried and jerky. Urine high coloured, acid, sp. gr. 1021, containing no albumen or sugar.

He was put on simple diet with milk and beef tea. An aperient was ordered, and carbonate of ammonia 4 grains and tincture of digitalis 10 minims were given every four hours. His general condition improved rapidly.

On the 19th the pulse was 120, weak, soft, and regular; the respirations 40.

On the 22nd.—Pulse 96, soft, but more full and slightly irregular; resp. 28.

On Nov. 3rd the pulse had fallen to 60.

My own interviews with the patient were so perfectly similar that the indications obtained may be summarised.

The speech had the same peculiarities throughout. When asked a question he would make a brief reply as if he understood and answered; the modulation of the voice and the emphasis were perfectly natural, and corresponded with the facial expression and gestures, but as a rule there was not the least semblance to words in what he said. He never even gave a simple yes or no. He would often address a long story to us, sometimes argumentative in tone, with much appropriate gesture and corresponding changes of facial expression; or he would obviously be making some request, and would plead with great earnestness, going on to

crying and beseeching. We sometimes guessed that he wanted alcohol, and once he apparently wished to leave the hospital. It was when he was excited and emotional that words and phrases would slip out in the torrent of inarticulate sounds, such as "If you please," "Thank you—thank you."

It was a matter of great interest and importance in the estimation of the mental condition of the patient, and of the loss of intellectual faculties he had sustained, to make out how far he understood what was said to him. His replies were often so suitable in length and emphasis that had there been no other means of forming a judgment it must have been supposed that he had comprehended the question. When, however, he was told to do anything it was seen that he did not understand the simplest phrase. He sat up in bed once or twice when required to do so, but as this was not made a test question, there would be other indications of what was wanted, and he was extremely ready in comprehending signs. When told to give his hand he invariably put out the tongue, and would do this several times during the same visit. When told to shut his eyes he sometimes obeyed and sometimes did not, but whether he closed the eyes or not, he put out his tongue.

The direct question whether he would like any of the intoxicating drinks he was known to be fond of was avoided, as there was no intention of allowing him any, and it would have been cruel to tantalise him, but when they were mentioned in his presence, or when allusion was made to his habits, there was no indication that he understood what was said; the opportunity was not seized of manifesting his desire when the name was mentioned, as in ordinary aphasia. On one occasion only, when the sister asked him what he had had for tea, he is said to have answered "Bread and butter."

Other interesting particulars were obtained by Mr. M. Handfield Jones. It has already been stated that sensation was found by him to be impaired in the right half of the body and limbs. At almost every visit the patient implored

him for something, and the word "Drink" could sometimes be made out; "If you please," "Thank you," also were distinguished; he frequently cried, but less after he had been in the hospital for ten or fourteen days than at first.

Mr. Jones one day handed him a letter addressed to him at the hospital; he took it, appeared to read the name and address, and then put it down. Not attempting to open it, Mr. Jones then wrote on a piece of paper "Give me your hand," and called the patient's attention to it; he took it, held it so as to get a good light on it, and then, having apparently read it, laid it aside without giving his hand, though asked to do so by word of mouth as well as in writing.

He lay in bed and manifested no desire to rise. He would sit up in bed and appear to watch with interest what was going on in the ward, but did not seek to enter into conversation with other patients. When the meals were brought in he looked for his, and ate his food naturally. When the bowels were about to act he called the attention of the nurse by knocking on his locker, and then pointed to the commode. There was never anything extraordinary in his behaviour.

On Nov. 3rd he was got out of bed and dressed. He walked with rather a tottering gait to the fire and sat there for a short time; after which he rose from the chair, walked back to bed, and got in with his clothes on.

His death was sudden and unexpected. During the night, Nov. 5th and 6th, the resident medical officer was called to him. Before his arrival the patient had got out of bed and taken a large dose of his medicine (about 30 minims of Tinct. Digitalis); he had then tried to reach the stool, but could not. He was found in a state of extreme dyspnoea, and blue and livid in the face. Artificial respiration was tried, but he died in a very short time asphyxiated.

Post-mortem examination.—The head only was allowed to be examined. When the skull-cap and dura mater, which presented nothing remarkable, had been removed, the pia mater was seen to be white and opaque over the convexity

of the hemispheres generally, and to be raised from the convolutions by a considerable amount of fluid. This gave an opalescent appearance to the convexity of the brain. On careful examination a yellow discoloration could be seen through the membranes and fluid at the situation of the postero-parietal lobule near the longitudinal fissure, and could be traced downwards towards the posterior end of the fissure of Sylvius. This part was also soft to the touch.

When the brain was removed from the skull it was found to weigh 47 oz. The vessels at the base were almost free from disease. The membranes here were transparent, and when the fissures of Sylvius were explored nothing abnormal was detected till the extreme end of that on the left side was reached, and here the middle cerebral artery was found to be occluded by a fibrinous plug.

The two hemispheres being separated it was seen that the ventricles were in a slight degree distended by clear fluid, but all the parts seen in them were normal, and the septum lucidum intact.

The right hemisphere, except that the membranes on the convexity were opaque, and that there had been fluid beneath them, was sound.

The left hemisphere presented obvious disease, limited to its posterior half. The frontal and ascending parietal convolutions and the anterior part of the temporo-sphenoidal lobe were healthy in appearance and consistence.

Above the posterior end of the fissure of Sylvius the convolution forming the supra-marginal lobule was yellow in colour, shrunken in volume, and soft. This condition extended upwards and backwards to within about half an inch of the longitudinal fissure just in front of the external parieto-occipital fissure, involving, therefore, the postero-parietal lobule. Extending backwards the morbid change implicated the angular gyrus, and nearly reached the occipital lobe; in a downward direction the adjacent parts of the temporo-sphenoidal lobe, the posterior end of the infra-marginal, and parallel gyri, were soft, but not wasted or discoloured on the surface.

(1) Examined by a succession of vertical transverse sections from the frontal extremity of the hemisphere backwards, softening was first encountered in the lower end or foot of the posterior ascending parietal convolution, quite at its posterior part; it did not here extend into the fissure of Sylvius, and there was no external discoloration. On the lower side of the fissure in the temporo-sphenoidal lobe, there was softening in the infra-marginal gyrus to a depth of half an inch. (2) Half an inch further back, and about three quarters of an inch from the end of the fissure of Sylvius, the section found the substance of the supra-marginal lobule soft and almost diffuent, and its surface in the fissure of a brownish-yellow colour; the temporo-sphenoidal lobe was also softened nearly to its inferior surface. (3) A section across the extremity of the Sylvian fissure passed through the maximum of softening, which here extended from near the longitudinal fissure, across the convexity of the hemisphere, to within half an inch of the inner and inferior border of the temporo-sphenoidal lobe; the greatest depth of the softening was midway, and measured about three quarters of an inch. In successive sections behind this point, the area of softening became narrower and its depth less; it extended backwards midway between the upper and lower borders to within an inch of the occipital extremity, being very shallow at its posterior part. It involved of course the angular gyrus and the middle *pli de passage*. The softening did not at any part reach the central ganglia or the ventricle.

The part of the hemisphere affected was thus the regions supplied by the third and fourth branches of the middle cerebral artery, and the convolutions softened were the postero-parietal lobule, incompletely and superficially; the supra-marginal lobule throughout its extent, and in depth down to the oval white centre; the entire angular gyrus profoundly; the annectent gyrus behind it superficially; the posterior half of the infra-marginal or first temporo-sphenoidal gyrus; and opposite the termination of the fissure of Sylvius almost the entire thickness of the temporo-sphenoidal lobe. The frontal lobe and the oblique ascending

parietal convolutions, in which the motor centres have been localised, were, as has been already said, free from disease.

Whatever may ultimately be the interpretation placed upon them, cases like this are worthy of careful study. A definite lesion is found, to which, from the history of the attack, there can be no hesitation in assigning the symptoms. It is important, therefore, that a definite idea should be formed as to the nature and extent of the loss of function sustained. Under each of the general descriptive terms in common use, such as coma, delirium, unconsciousness, insensibility, are included conditions essentially different, and an analysis of them may be as fruitful of results as a "study of convulsions" has proved to be in the hands of Dr. Hughlings Jackson. Even words like aphasia and amnesia, brought into use for the express purpose of defining a single invariable condition, become snares and hindrances by being employed to designate by the same name affections which a careful description would have shown to be different.

The patient in the case under consideration had lost the faculty of understanding words spoken or written by himself or others, so much so that he was not even aware that his own attempts to talk were simple gibberish; the question is whether this alone would account for his condition, or whether there was not some further and more general impairment of the mental powers. It was of course impossible to test his memory, but he recognised his friends, and knew what he might expect from his medical attendants and from the nurses, addressing to the former his inarticulate requests, and summoning the latter when he required their assistance. He manifested great disappointment also when any resident medical officer or clerk passed through the ward without noticing him. Again, he had obviously ideas in his own mind to which he thought he was giving expression, and his gestures and changes of facial expression were remarkably significant and appropriate. On superficial observation he might have been set down as simply imbecile or demented, but a far greater degree of imbecility is seen without any approach to his want of comprehension of words or to the

inarticulate character of his speech, and it is not difficult to conceive the helplessness and bewilderment of a man who suddenly finds himself unable to understand what is said, and sees that those around him fail to understand him, while he supposes himself to be speaking as well as ever. Had the patient been an Asiatic, and his only language one of which we were absolutely ignorant, we should not have discovered that he was suffering from any impairment of the mental faculties.

It seems probable, then, that the complete loss of comprehension of words exhibited by this patient would account for his mental condition, and the interpretation here placed upon this loss is that it was due to destructive lesion of an area of the cerebral cortex, in which perceptions derived through the different senses are combined and elaborated into ideas of the objective causes of sensation, which ideas, again, are in this centre associated with and symbolised by names or words. If the interpretation is correct, we have at some part of the cortex involved in the softening the centre in which "concepts" are formed, "naming" takes place.

It may be considered fortunate that the problem was not complicated by extension of the softening to the third frontal convolution. Had this been the case it would have been impossible to exclude the hypothesis that the amnesia was represented structurally by lesion involving the same cortical area as aphasia, but more extensive, and that thus amnesia was an aggravation of aphasia. There being, however, no lesion to which aphasia could be attributed, the distinction, arrived at by analysis, between words considered as intellectual symbols, the elaborated product of sensory impressions, and words considered as motor processes, is confirmed by disease. Dr. Hughlings Jackson has aptly called the third left frontal gyrus the "way out" for words; in the case related it was the "way in," and the centre in which words are formed as "concepts," which were damaged or destroyed.

It is important to note the fact that an affection of language other than aphasia is produced by disease in the *left* hemisphere. The softening was extensive, including the auditory perceptive centre of Ferrier or a part of it (the posterior half

of the infra-marginal gyrus), the visual perceptive centre (angular gyrus), as well as the more hypothetical centre for concepts and names not yet localised; but affecting one hemisphere only the observed results go beyond the anticipations we should have formed. Still more remarkable is the case mentioned in which a limited lesion in the white substance underlying the angular gyrus of the left hemisphere, and presumably isolating it from this centre for concepts and names, abolished the power of naming an object at sight. With regard to these, as in aphasia, there arises the question—What would be the effect of a similar lesion in the opposite hemisphere? Destruction of the third frontal convolution on the left side of the brain gives rise to aphasia; on the right side it does not. The precise results of lesion of the right third frontal gyrus, if not simply negative, are not definitely known. Apparently the employment of this convolution in the left hemisphere as the “way out” for language involves the predominant, if not exclusive, employment of this hemisphere at other stages in the complex process by which language becomes the vehicle or, it might almost be said, the instrument of thought. This, again, according to the hypothesis of Broca and Moxon, is a secondary consequence of right handedness, the dextral pre-eminence of the hand implying sinistral pre-eminence of the brain. It may already be taken as established that there is a marked functional difference between the two hemispheres, which in structure and arrangement are so nearly alike, but whether this consists simply in a relative superiority of the left, or whether there is thrown upon the right some compensatory superiority in a less conspicuous function than that of language, cannot yet, in the opinion of the writer, be determined. A comparison of the results of lesions in similar parts of the right and left hemispheres will afford information of extreme interest and value. To this Mr. Callender has made an important contribution in the ‘St. Bartholomew’s Hospital Reports’ vols. iii and v, 1867 and 1869. The question is, however, too large to be opened in the present communication.

AN ACCOUNT OF 500 CASES
OF
OPERATION FOR STONE IN THE BLADDER
OF THE MALE ADULT.

COMPRISING HIS ENTIRE EXPERIENCE OF SUCH CASES
TO JANUARY, 1877;

*With Remarks on the most important Incidents which have occurred in
connection with them.*

BY

SIR HENRY THOMPSON, F.R.C.S.,
SURGEON-EXTRAORDINARY TO H.M. THE KING OF THE BELGIANS; CONSULTING
SURGEON TO UNIVERSITY COLLEGE HOSPITAL; AND EMERITUS PROFESSOR
OF CLINICAL SURGERY AT UNIVERSITY COLLEGE, LONDON.

(Received January 8th—Read March 13th, 1878.)

I PROPOSE to record briefly the leading particulars of five hundred cases of operation for stone in the bladder, occurring in male patients only, of twenty years old and upwards. The calculi removed in all these cases are, with a few exceptions, also laid before you.

I have said that the patients were of "twenty years old and upwards;" but it is worthy of remark that there is one only of the age named, and only twelve patients in the entire list are below thirty years.

This group includes every adult male whom I have treated

either by lithotomy or by lithotripsy between September, 1857, when my first case occurred, and January, 1877. It comprehends therefore all my patients at University College Hospital and my private practice.

I purposely exclude all cases below twenty years of age, and all cases in the female sex at any age. I do so because the risk from stone operations on boys and women is exceedingly slight as compared with the risk in the adult male; and no true judgment can be formed in regard of the results of lithotomy and lithotripsy, if female cases and males at all ages are indiscriminately included in the same group. Thus, for example, during the whole of that period, I have met with but one death in a male below twenty years of age, and that in a boy with extreme deformity of the bones of the pelvis; and with but one in a woman, and that from pyæmia.

I wish first to refer to the experience of the chief operators before my time who have published their results. Not one in this country has had the good fortune to treat a number approaching that which furnishes the subject of this paper. And perhaps I may be permitted to add that there is no record with which I am acquainted that has been kept with such scrupulous care. For I possess full notes of every case from the first to the last, made at the time, in books arranged on a methodical plan at the outset, and continued without change of system to the present day.

The first trustworthy numerical record we possess is that by Cheselden. That famous surgeon, at the close of his career, reported as follows. Telling us first, "of his private practice he kept no account," he states that "he cut 213 cases at St. Thomas's Hospital," pertinently adding, "but what is of most consequence to be known is the ages of those who recovered and those who died." Of these 213 cases, no less than 135 were under ten years of age, and 3 died; 62 more were between ten and twenty years, and of these 4 died. *Only 46 were above twenty years of age, and of these 13 died.*¹

Long after this comes Martineau's remarkable and excep-

¹ Cheselden's 'Anatomy,' &c., 5th edit., London, 1740, p. 333.

tional series of successful cases, viz. 84 operations with 2 deaths; although this did not represent his entire experience, which I have heard at Norwich was by no means equal to the published cases, being indeed not above the usual average. Of those 84 cases, however, 6 were females, and 36 of the others were under twenty years of age, leaving 42 cases of twenty years and upwards, among which were the 2 deaths.¹

Sir Benjamin Brodie reported at the close of his career 115 cases of lithotrity in the adult male; adding, "I do not mean to say that these occurred in the same number of individuals," and mentioning one patient "on whom the operation was performed as many as eight times in the same number of years, always with complete success;" and also a second case resembling it. Among these were 9 fatal cases or "somewhat less than 1 in 12½."²

Sir William Fergusson in 1867 reported the total of his cases at that time as 271. Of these, 219 were adults (but certainly some were females), and 110 of them were treated by lithotomy, with 33 deaths; while 109 were treated by lithotrity with 12 deaths; total, 219 cases with 45 deaths, a mortality of adults treated by both methods, of 20 per cent. The remaining 52 cases were in children with 2 deaths; an illustration as Sir William also points out of the need to report them separately.³ I have already written to Mr. Henry Smith for any information he might possess relating to Sir William Fergusson's subsequent experience. He replies that he does not possess any, and believes that probably "his additional cases would not amount to very much."

The total number of adult cases reported by these four operators is 422 cases.

I will now proceed to analyse the 500 adult male cases before you. Four hundred and twenty-two were operations by lithotrity, and 78 were by lithotomy; the relative propor-

¹ 'Trans. of the Royal Medical and Chirurgical Society,' vol. xi, p. 402.

² Ibid., vol. xxxviii, p. 169.

³ 'Lectures on the Progress of Anatomy and Surgery, 1867.' By Sir Wm. Fergusson, Bart. London. P. 220.

tion being 2 cut for every 11 crushed, or 1 cut in every 6½ cases.

The mean age of the 500 cases is no less than sixty-one and a half years; the youngest being twenty years of age, the oldest eighty-four years.

The number of individuals is 440. Several of the lithotrity patients have been operated on twice; 5 as many as three times, and there is one very remarkable instance of four operations on the same individual. In all these cases of operation repeated on the same person, there has been a considerable lapse of time between each performance; and full evidence of the existence of a fresh formation has been present, or the case would not have been so registered.

The removal of some phosphatic concretions, a by no means infrequent circumstance, from the bladders of elderly men who do not empty the organ naturally, has never been admitted into the category of operations for stone. They form a class by themselves to be referred to hereafter, in which the lithotrite is of the highest value in preventing the formation of large phosphatic calculi. But in constitutions which are strongly disposed to excrete a large amount of uric acid by the kidney, it not infrequently happens when the first calculus has been removed, that after an interval of two or three years of freedom from all vesical symptoms, they again appear and a second formation is detected, and lithotrity is again resorted to. A third formation is not very common, nevertheless in exceptional instances the process may be even again repeated. Thus in the one case referred to, the patient formed three large uric acid calculi consecutively, with several years of good health between each, and finally, a large phosphatic one, and each of these was successfully removed by lithotrity. This is a history nearly unique in my experience, and those four large calculi may be seen in a group, all belonging to Case No. 62 in this series. They occurred in a gentleman who passed all his time in London, where he was well known, and who died at an advanced age some time after the last operation.

The nature of the calculi is a matter of interest, and not without importance.

Of the 500—

- 313 were uric acid and the urates, in the ordinary sense of the term; in all calculi there is usually found a slight admixture of other varieties.
- 99 were phosphatic of the ordinary kinds.
- 50 were mixed; urates and phosphates.
- 9 were mixed; urates and oxalates.
- 16 were oxalate of lime.
- 6 were mixed; oxalates and phosphates.
- 1 was cystic oxide.
- 1 was pure phosphate of lime.
- 1 was phosphate and carbonate of lime, probably formed on large nuclei from the prostate.
- 4 were phosphatic, deposited on foreign bodies introduced into the bladder; these being, in two cases, a portion of exfoliated bone; in one a piece of sealing wax, and in one a piece of bougie.

I have never considered the removal of a foreign body from the bladder as equivalent to an operation for stone, unless the body has been really the nucleus of a calculous formation. Thus a hair pin recently introduced into the male bladder and successfully removed by a lithotrite is not admitted into the series, as it cannot be regarded as illustrating in any way the subject before us, viz. operations for stone. Furthermore, the small proportion of phosphatic calculi in the above list—less than the recognised average proportion—will illustrate a foregoing remark, that mere phosphatic concretions and deposits have been similarly rejected from the list.

I desire next to refer to the difference in the relative proportion in which the two operations have been employed in the series before us, as compared with that which has heretofore obtained in this country. For example, Sir William Fergusson's cases of lithotrity were just equal in number to his cases of lithotomy, that is to say, 109 against 109. This result partly due to the fact that he performed a good number of the latter before lithotrity had become an accepted country. Reckoning from the time at

which he adopted the method by crushing, his numerical proportion was probably at least 3 cases of lithotritry to 1 of lithotomy. But in my series the crushing operation has held a ratio in proportion of $5\frac{1}{2}$ cases to 1 of lithotomy. Throughout the entire term the small stones and the most promising cases have been reserved for lithotritry; while, as the necessary consequence, the knife has been employed only for cases in which the stone has been large, or when it has been associated with obstacles which render lithotritry difficult or impossible. As certainly during my life I have never rejected more than 5 cases, if indeed so many, as unfit for any operation, it follows that the most unpromising and difficult cases fell into the category for which the cutting operation was reserved.

I shall next take the question of mortality following the operation; first, in relation to lithotritry (see Table I); secondly, in relation to lithotomy (see Table II); and lastly, in the aggregate as affecting the entire 500 cases (see Table III).

TABLE I.—*Deaths following Lithotritry.*

Age.	Remarks.	Cause.
68	Unfit for operation	Calculus disease of kidneys and dilated ureters.
69	Diseased kidneys and dilated ureters.
81	Unfit for operation, if any he should have been cut	Hæmorrhage.
69	Fever, mania, delirium tremens(?).
77	Unfit for operation, treated at a distance in the country	Acute cystitis and fever.
64	Disease of heart	Exhaustion.
69	Unfit for operation, but demanding relief	Ditto.
73	Ought to have been cut, but he refused	Acute cystitis and fever.
80	Fever and exhaustion.
59	Pyæmia and local abscesses.
68	Ought to have been cut, but he refused	Acute cystitis and fever.
70	Nephritis and suppression of urine.
72	Stone, large, might have been cut	Chronic Bright's disease.
65	Ditto	Acute nephritis.

Age.	Remarks.	Cause.
62	Acute cystitis and fever.
55	Chronic Bright's disease and pyelitis.
62	Pyæmia, joint affections.
74	The pyæmia occurred after he left town, 6 weeks after operation	Phlebitis of leg after operation, and subsequent pyæmia.
32	Unfit for operation, which was done to relieve suffering	Chronic Bright's disease.
64	Pyelitis and exhaustion.
52	Pyelitis and fever.
66	Probably might have been cut	Diseased kidneys and dilated ureters.
77	Pyæmia.
75	Very feeble and unfit for any operation	Fever and exhaustion.
57	Large stone, very feeble	Exhaustion.
65	Diseased kidneys and dilated ureters.
66	Ditto.
81	Fever and exhaustion.
61	Diseased kidneys and dilated ureters.
70	Pyæmia.
58	Nephritis and exhaustion.
75	Fever and exhaustion.

TABLE II.—Deaths following Lithotomy.

Age.	Remarks.	Cause.
74	10th day after	Exhaustion.
68	21st "	Ditto (P).
73	5th "	Ditto.
74	3rd "	Peritonitis.
73	23rd "	Exhaustion.
80	24th "	Ditto.
69	14th "	Diseased kidneys and dilated ureters.
59	3rd "	Peritonitis, perforation by staff.
78	11th "	Secondary hæmorrhage (at a distance).
67	22nd "	Chronic peritonitis, sacculated bladder.
50	3rd "	Peritonitis.
79	4th "	Exhaustion.
65	5th "	Peritonitis, diseased kidneys, and dilated ureters.

Age.	Remarks.	Cause.
68	4th day after	Nephritis and suppression of urine, enormous prostate.
29	35th " certainly not a death from operation	Chiefly from bedsores producing exhaustion.
68	3rd "	Peritonitis.
55	12th "	Secondary hæmorrhage.
36	14th "	Diseased kidneys and dilated ureters.
65	7th " to relieve great suffering	Exhaustion.
60	3rd "	Peritonitis.
82	18th "	Exhaustion.
60	7th "	Peritonitis from sac of bladder suppurating.
47	3rd " any operation of questionable utility	Chronic Bright's disease and exhaustion.
70	3rd "	Hæmorrhage.
78	12th "	Exhaustion.
61	2nd "	Peritonitis, perforation of bladder.
61	4th "	Nephritis and suppression of urine.
79	5th " operation undesirable, to relieve great suffering	Exhaustion.
72	7th "	Pyæmia.

TABLE III.—*Causes of Death in 500 Cases of Operation for Stone.*

	Disease.	Lithotrity.	Lithotomy.
1	Pyæmia	5	1
2	Diseased kidneys and dilated ureters	6	2
3	Acute nephritis	3	2
4	Chronic Bright's disease	3	1
5	Pyelitis and fever	2	—
6	Peritonitis, large sacs of the bladder in two cases	—	7
7	Perforation of bladder	—	2
8	Acute cystitis and fever	4	—
9	Exhaustion	7	11
10	Hæmorrhage	1	3
11	Mania	1	—
	Total	32	29
		61	

And here it is necessary to say that I have frankly accepted any death occurring within six weeks after the operation, under almost any circumstances, as a fatal issue to be reckoned to the tale of mortality. Thus, one of the deaths from lithotrity by pyæmia occurred six weeks after the operation, when the patient had left town, which he did with phlebitis in one leg. The veins of the leg had no doubt been affected through the circulation by the inflamed vesical veins, and thus pyæmic infection of the system resulted. Again, I left, one summer holiday, at the hospital a case of lithotomy, three weeks after operation, with the fullest assurance that he would recover. He had then a small bed-sore. On my return two months subsequently, I learned that he had died a fortnight after my departure from bed-sores and exhaustion, and I thought the death ought not to reckon to the operation. Both the patients I have nevertheless accepted in my list of fatal cases. There are three instances, however, among this large number of men at advanced age, of death occurring suddenly from organic disease of the heart, which was in no way connected with the process adopted for the removal of the calculus. The history of every case is given in a voluminous table presented with this paper; from which it may be seen that in one, a man, apparently in excellent health, undergoing lithotrity with little inconvenience, falls dead when at the water-closet, and is found at the autopsy to be the subject of old mitral disease. Two others were similar to a greater or less extent. Desiring then to deal with the group so as to arrive at a fair estimate of the mortality, all other deaths but these three cases have been included in it. Some remarks on the causes of death will follow hereafter.

These conditions considered, I come now to the numerical proportion of fatal cases.

In the 422 cases of lithotrity there were 32 deaths; or a mortality of $7\frac{1}{2}$ per cent., or 1 death in rather more than 13 cases.

In the 78 cases of lithotomy there were 29 deaths, or about 1 death in $2\frac{3}{4}$ cases.

Taken collectively, in the 500 adult cases treated by both operations, there were 61 deaths, or a total mortality of 12 per cent., or 1 death in $8\frac{1}{2}$ cases.

Let us briefly compare this result with the mortality recorded as the result of lithotomy and lithotripsy by the operators already named.

Cheselden's cases of adults over 20 years—46, with 13 deaths.

Martineau's	"	"	42,	"	2	"
Brodie's ¹	"	"	115,	"	9	"
Fergusson's	"	"	219,	"	45	"

Making a total of adults . 422, " 69 "

The mortality of the conjoint cases is within a fraction of $16\frac{1}{2}$ per cent., or 1 death in slightly more than 6 cases.

I will now refer to a table of lithotomy cases only, of all ages (which I obtained by great labour, some years ago, for my work on 'Lithotomy and Lithotripsy') by the leading hospital surgeons of Great Britain, from whom I obtained written records of each individual patient, amounting to a total of 1827 cases. Among these 1827, there are 736 cases of twenty years old and upwards, with 149 deaths, or slightly more than 1 death in 5 cases. This result shows a remarkable similarity to the table now made of Cheselden's, Martineau's, Brodie's, and Fergusson's operations.

I think I may venture to say that the 12 per cent, or 1 death in $8\frac{1}{2}$ adult cases, which belongs to the series before you, is the best result which has yet been shown in any authentic report of any man's *entire and unselected work*. And I will add that I believe it to be greatly due, among other things, to the care taken in deciding which of the two operations best meets the individual needs of each case.

I have long ago said that in accuracy of diagnosis, and in the proper selection of the operation which must follow, is to be found the key to successful results; and I have taken

¹ It is to be borne in mind that Brodie's were cases selected for lithotripsy, and that we know nothing of the result from the lithotomy performed or the cases which he rejected as too serious for the crushing operation. This gives a manifest advantage to the table above. So also does the incorporation in it of Martineau's selected series.

pains to show that without proper diagnosis in relation to the size of the stone and the condition of the patient (the former point being one of extreme importance), *it is more dangerous to employ the two operations than to practise lithotomy uniformly in all*. For unquestionably lithotripsy is incomparable for safety when the stone is of moderate size, but is inadequate and dangerous for large ones; for these a well performed lithotomy is a less severe proceeding, while it is too severe and dangerous for a small stone.

I wish very briefly to note a few particulars which my experience has afforded, and which may be interesting to others. It will be manifest that space forbids me to touch on the ordinary points; reference will therefore be made to extraordinary occurrences.

The first section will relate to the accidents which I have met with in the performance of the two operations, and to the causes of death. The second will relate to the after history of patients who have been submitted to lithotripsy.

First, accidents in operating. In performing lithotripsy I have on one occasion broken a lithotrite in the bladder. In this case, a patient æt. 64, the stone was one which I did not consider ought to have taxed the power of the instrument unduly. What happened was this; while in the act of crushing, the end of the male blade snapped off close to its junction with the shaft, so that a slip of steel three quarters of an inch long remained free in the bladder; I withdrew the instrument and cut the patient next day, removing the stone and the broken piece, and he made a good recovery. I might easily have withdrawn the piece of metal by the lithotrite, but the texture of the stone (although not oxalate of lime) decided me to cut the patient. The case is No. 224 in the series. I may remark that the fracture took place precisely where, if fracture is to happen, it ought to occur. It was an old and very favourite lithotrite, and had been used for about 150 cases, which is quite as much work as any lithotrite is capable of safely performing. Long ago, in describing the construction of the instrument, I stated what Charrière, of Paris, had taught me, namely, that the parti-

cular point described ought to be the weakest, as a fracture there is really of little consequence, and prevents a twist or fracture elsewhere if undue force happens to be used.

In a second case I had a piece of uric-acid calculus not large, but so impacted between two flat blades, that I could neither get rid of it nor could I withdraw the instrument. In these circumstances I accomplished what I think has not been precisely thus done before; I very slowly drew the instrument and its contents through the neck of the bladder and the prostatic urethra, which are remarkably dilatable if only time is given to the process, and steady traction is performed coolly and without hurry. Thus it came still further forward, and when the urethra ceased to yield any more, I pushed the testicles backwards and made a short incision in the median line of the penis towards its root down to the urethra, until I reached the lithotrite, the end of which was then easily pushed into view through the opening, when the débris which had been impacted were removed, the blades closed, the instrument returned through the wound to the urethra, and withdrawn as usual by the external meatus. There was a little extravasation next day requiring one incision in the scrotum; all, however, soon healed; the patient recovered and is perfectly well at this day. This accident happened in 1874, Mr. Clover gave the patient chloroform and witnessed the whole proceeding; the case is No. 204. I have had no other accident in 422 cases of lithotrity. There was one death from hæmorrhage in a patient æt. 84, who had a considerable quantity of hard stone in his bladder. This was a very early case, No. 13, and one in which I should not now entertain the idea of adopting the crushing operation.

I now come to accidents occurring in lithotomy. In this operation I have met with a cause of death on two occasions, viz. perforation of the bladder by the staff. I confess that the circumstance was new to me, and I think it deserves careful attention. Both cases occurred some time ago at the hospital, and were publicly examined in the deadhouse there. I had often observed before the time of these accidents, the

extreme tenuity, and in some cases, softness, of the vesical coats in disease. I have examined bladders at a very early period after death, and before much post-mortem change can have taken place, so soft as to tear almost like wet paper. Now nothing can be more likely to happen sometimes, unless we are on our guard as to this source of danger, than the pushing the point of a staff through the upper fundus of a contracted bladder, if the instrument be so long as it often is. I have since been frequently struck with the unnecessarily long curve which some staffs have, and have felt, when seeing such an one in use, how dangerous its point must be in a bladder affected in the manner described. I am strongly inclined to think that in those cases of rapidly fatal peritonitis which are sometimes met with after lithotomy, if the search were carefully made, a minute rupture at the upper and back part of the bladder would be sometimes found. It may be very small, lying between the rugæ of the organ, and easily escape observation, yet give issue to a small quantity of urine into the peritoneal cavity; for it is not very easy to explain otherwise some of these attacks which carry off a patient in thirty-six hours or so after operation. Extension of inflammation from the bladder to its peritoneal covering has been suggested, but I think the mechanical cause a more probable one. Let me say that injury to such bladders may as readily be inflicted by the forceps as by the staff, but I do not think the knife has any thing to do with it. Clearly, in the two cases in question, the little openings were not so made; both the character of the orifice and its situation preclude such an explanation. Besides, I am quite sure that the danger of perforating the bladder with the point of the knife must be an extremely remote one, and impossible in fairly competent hands.

Secondly, secondary hæmorrhage has been fatal in three cases, that is to say, the bleeding having perhaps been free at the operation, but controlled, and ceasing altogether for a week or more, a sudden outbreak has occurred on the ninth or tenth day after, and the patient has succumbed. I have recently met with this occurrence on the fifteenth day when

the patient's life was in great jeopardy, but it was stopped by introducing the "tube en chemise" and stuffing it well, by which process the half-healed wound was wholly reopened. He made a slow but good recovery, and is now quite well. I have no doubt that the process of healing in arteries divided in elderly patients, is less rapid and perfect than in those of others, and that incisions in men between sixty and eighty years, as most of these were, involve risk from both primary and secondary hæmorrhage not met with in young and middle aged persons. But the occurrence of hæmorrhage, whether primary or secondary, I am happy to say I no longer dread. Thanks to a simple contrivance of my assistant, Mr. Buckston Browne, the india-rubber "tampon," inflated with air, there need be no uneasiness in future on the ground of hæmorrhage, since it may be completely commanded by this little apparatus. No man ought henceforth to think of cutting a patient without one at his side.

Another accident, but not a fatal one, which I have to name, is wounding the rectum. I confess that I am not surprised that it occasionally happens in those cases of elderly men in whom the rectum is sometimes a remarkably capacious cavity, and is felt when explored by the finger, surrounding partially, as it were, the prostrate and neck of the bladder. I think a certain proportion of such accidents must be met with by any man who operates frequently on patients of sixty years old and upwards. No doubt the danger of injuring the bowel is greater when "cutting low," as it is termed, that is by entering the point of the knife into the groove of the staff rather low down or far back. On the other hand by "cutting high" or entering the staff easily where it is not so far from the surface, the rectum will be perfectly safe, but the bulb or even its artery may be divided and the occurrence of severe hæmorrhage is risked. These two dangers are the Scylla and Charybdis of the lithotomist when dealing with old people. In youth and middle life there is less fear of injuring the rectum, and a lower incision may be made. I have wounded the rectum four times, and in the last case I laid open the incision into the t-

month afterwards, for I was not aware of the injury at the time of operation, indeed, there was no sign of it until a week after, and it is possible, although I do not take advantage of that supposition, that I may have left a very thin partition between the wound and the cavity of the bowel which subsequently sloughed. The patient was sixty-four years old, and the stone was a rough and spiked example of oxalate of lime. It is the only case in which I have divided the septum between the wound and the bowel, and it is the only case in which I have had any serious trouble subsequently, and the condition still exists and is a cause of great discomfort. In one of the previous instances death occurred by pyæmia, and in the second and third the opening was healed in one case, and in the other nearly so, by the electric cautery, which by the way has been of little service in the more recent case above referred to.¹

I may next say, before leaving the subject of lithotomy that in the earlier part of the series, I was anxious to test the various median operations which had been then much discussed; and that 20 out of the first 27 operations were those known as the median, medio-bilateral, and the bilateral of Dupuytren. After these I returned to the lateral operation, and the mortality has gradually diminished ever since; the first third of the 78 cases showing fewer deaths than the second third, and the latter third less than the second third; the last 20 having indeed been followed by only three deaths. This result is no doubt in part due to increased experience of the operation, but in part also to the superiority, as it appears to me, of the lateral to median operations for cases of rather large stone. For it is clear that lithotomy is now demanded only for such cases, lithotripsy being competent to the successful management of small ones, and of this I think the proof is before us.

I propose next to make a brief analysis of the causes of death in the total of 61 cases, as presented in the table of

¹ Since writing the above I have received a letter from the gentleman, dated January 2nd, 1878, informing me of the steady improvement which has of late been taking place in his condition.

two columns (see page 166). Those causes are of course similar for the two operations in cases of advanced disease of the urinary organs existing at the time; a condition unhappily frequent among patients who have been long the subjects of calculous disease. But each operation has also its special risks and special causes of death. Regarding first the causes of death common to the two operations, it will appear that that disease of the kidneys, with distended pelvis and dilated ureters, which seems mainly due to mechanical obstruction below, was found by autopsy in eight cases; it probably existed in some others not examined. Under the head of exhaustion, no less than 18 cases are set down, respecting which it is necessary to say that by this term is intended that failure of power at advanced age, which—no very obvious clinical sign of constitutional disturbance or of local disease existing—more or less gradually issues in dissolution. This group includes cases in which no autopsy could be made, and there is little doubt that in many of them, serious changes in the kidneys and ureters were taking place. Of these cases 7 belong to the lithotripsy column, and 11 to that of lithotomy. But acute nephritis, pyelitis, and cystitis, are found mainly among the cases of lithotripsy. On the other hand, 7 cases of peritonitis, besides the two caused by perforation already referred to, occurred after lithotomy only, I have no doubt that this cause of death may be rendered less frequent by increased care in the management of instruments in the bladder. In 2 of the 7 cases large sacculation of the bladder existed, and the sacs were inflamed and suppurating; an autopsy was not obtainable in every case, and this condition, always a most dangerous one in connection with either operation, was probably present in another case or two. Chronic Bright's disease was present in 4 cases. Fatal hæmorrhage, as already noted, was present also in 4.

The occurrence of pyæmia demands a remark. In the 32 deaths following lithotripsy, 5 were due to this cause. The connection between it and the operation is not difficult to be traced. Cystitis produced by the presence of sharp frag-

ments may produce inflamed vesical veins; and the products of inflammation in the circulation lead to infection of the system. In the 29 deaths following lithotomy, only 1 was due to blood poisoning, a fact which furnishes a striking illustration, if one were needed, of the small liability to infection possessed by wounds exposed to septic influences. The wound in lithotomy can scarcely be guarded by any arrangement which could be devised, from access of common air, nor from much that is manifestly impure, and no attempt has been made in these cases to prevent such access; yet with a prolonged contaminated exposure, often amounting to some weeks, only one instance occurred.

I will now make a few remarks on the after history of elderly patients who have been submitted to lithotrity. It has been said, and not without reason, that their condition subsequent to the operation is often unsatisfactory, and that many suffer from troubles which rarely occur to patients whose calculi have been removed by lithotomy. I am quite willing to admit that facts support this allegation to a considerable extent. As a rule the patient who has been cut and who has recovered, does not suffer again from vesical troubles, unless he forms another calculus, as he sometimes does. On the other hand many who have submitted to lithotrity become afterwards the subjects of chronic cystitis, and phosphatic deposits, sometimes to a distressing extent. This condition I have made the subject of careful investigation, and I am bound to say that while it may be generally avoided, I do not think it always can be so; and the occasional liability to troublesome chronic cystitis after lithotrity, must be accepted as part of the price to be paid for avoidance of the greater risk to life which lithotomy involves. But I have no doubt as to the cause of the condition in most instances, nor as to the fact that much may be done to mitigate that cause.

The first circumstance which gives rise to it is the long period of continuous injury to, and consequent inflammation induced in, the mucous coat of the bladder, by prolonged contact with the fragments produced in the operation of

crushing. If the series of sittings necessary to remove a stone, extends over six or eight weeks, or even longer, as I have often known to happen in years gone by, a condition of chronic cystitis may be set up, from which the bladder will rarely quite recover. Hence rapid progress is an essential element in the achievement of successful lithotrity. One sitting should follow another generally after an interval of one or two, or, at most, of three or four days, and as rarely more than four sittings are required, a fortnight ought to suffice in almost every case, for the removal of a stone of moderate size. And here may be noted a very important fact, viz. whatever amount of cystitis occurs after the first sitting, it is beyond all question the most efficient treatment, at once to crush again and remove the mechanical irritant without delay. Mr. Clover's apparatus for removing fragments by "aspiration," I regard as particularly useful in these circumstances, and in most cases it is a valuable adjunct to lithotrity. There is little motive therefore for extending the duration of the proceedings beyond the period named; recurring attacks of severe rigors and severe orchitis only compelling the operator to do so. So that if the patient has little or no trouble to contend with, an interval of one or two days between each sitting is ample, and if cystitis is set up, another sitting and not delay is the best treatment.

Secondly, at the end of the operation or during the term of convalescence chronic retention of urine not unfrequently occurs. By small degrees the bladder ceases to empty itself completely, until it always retains perhaps two or three ounces, or less, after each act of micturition; and this habit becomes confirmed if the catheter is not used as often as may be necessary to empty the organ as soon as the retention, however slight it be, is discovered. It is impossible to be too watchful, or too suspicious of the first approach of this state. Space forbids me to enter upon important details in relation to these two points, which I have, moreover, considered elsewhere, but I wish to advert to the intimate relation in which they stand to the after troubles

of lithotrity, and to add that when cystitis and chronic retention have been promptly dealt with at first, there will rarely be serious or long continued troubles following the operation.

One great value of the lithotrite should be mentioned here, viz. its capability of removing phosphatic deposits from the bladders of elderly men who are compelled to use the catheter to withdraw the urine habitually. It is very common for such to form phosphatic deposits in the bladder, which must become large masses or considerable calculi, if not removed while small by the lithotrite. This I have done for patients some scores of times, but never regard it as a case of lithotrity, unless the calculous formation is at least as large as a nut, and is of hard consistence. Nevertheless, before the time of lithotrity, each one of these formations must inevitably have increased until a calculus was formed, and was then removed by lithotomy or permitted to remain, in which case a miserable death certainly followed. If the function of the lithotrite were limited to the relief of such cases merely, it would still have been a most valuable addition to the resources of the surgical art.

The last practical observation which I will venture to offer regards the relative proportion, among stone cases of average gravity, which should be allotted to each operation respectively. With my present experience, I think, had I again to operate in these 500 cases, I should treat a rather larger proportion than I have done by lithotomy, certainly among my last 100 cases, I have thought it right to assign more to the cutting operation than in the previous 100, and the result makes me satisfied with having done so. The limits of applicability in regard to the two operations, which after all cannot be defined by absolute formulæ, may, I think, be approximately estimated in the following manner. :—

Lithotrity is almost certainly successful in a fairly healthy patient, if the stone is rather small, say no larger than a filbert of moderate size, when it can be disposed of in two, or at most three sittings. In upwards of 70 cases of calculus no larger than a nut, chiefly uric acid, I have had 1 death

only, and that from pyæmia. I think the extreme limit of applicability may be thus stated in relation to the crushing operation, viz. never employ it for a calculus which cannot be easily disposed of by a flat bladed lithotrite of English construction, and dispense altogether with the old instrument with fenestrated blades; I have not used one of the last-named lithotrites for ten years; they are doubtless more powerful, and they certainly are more dangerous instruments than the flat-bladed ones. When a stone requires one of these powerful instruments to crush it, be assured lithotomy will be a safer proceeding.

The question then naturally follows: with what sort of stone is the English flat-bladed instrument qualified to deal? I should reply: for a uric acid stone, the long diameter ought rarely to exceed one inch or at most an inch and a quarter, about the size of an almond in its shell; for a phosphatic and mixed calculus the size may be much more considerable; a stone of oxalate of lime should not exceed an inch. Circumstances of course may demand some modification of these rules—for instance, a tyro in the use of the lithotrite should not readily undertake to deal with a rather large example, which nevertheless might be safely crushed by a surgeon of experience.

In bringing this paper to a conclusion, I desire to say that there are some other points of interest which might be named, but the foregoing appear to me to be the most important. It has been my aim to compress within the narrowest limits a faithful account of the most important results of my entire personal experience. It might have been wiser—and it certainly would have been an easier, and to myself, a more satisfactory task, to present this report in three papers instead of in one. I decided, however, on accomplishing the latter object if possible, and none can better know than myself how imperfectly it has been obtained; but I believe that it is desirable in these busy days to err rather on the side of brevity than on that of prolixity.

ON THE
CONDITION OF THE SKIN IN TINEA
TONSURANS.

BY
GEORGE THIN, M.D.

(Received January 8th—Read March 26th, 1878.)

ALTHOUGH the discovery by Malmsten of the trichophyton tonsurans is now more than thirty years old, there are some essential points relating to the growth of the parasite in the skin that are still unsettled. Küchenmeister, for example, in his classical work 'On the Parasites found in and on the Human Body,' published in 1855, states that the parasite in question can only live and increase in the substance of the hair shaft itself, and in the crusts which are found on the scalp, and that it is never found between the cells of the epidermis. Kaposi, on the other hand, in the chapter devoted to herpes tonsurans in Hebra's work 'On Skin Diseases,' states that similar fungous elements (conidia and mycelium) to those found in the hair shaft are also found between the cells of the root sheath. The subject thus raised by Küchenmeister and Kaposi deserves the special attention of pathologists at the present time. The solution

of questions of the highest importance in medicine are now waiting on information which is being diligently sought for on many sides, regarding the conditions in which low vegetable organisms can live and multiply in animal tissues.

Anything which can be learned about the position of the spores in ringworm, has therefore a general, as well as a special interest.

If Küchenmeister is right, then in this disease the fungus can only live on dead animal products, for such the hair shaft and the desquamated cuticle, which he designates as crusts, must be considered; if Kaposi is right, then the fungus lives and propagates in the midst of living tissue.

The observations that have been made hitherto in regard to the subject, have been limited to an examination of hairs extracted from parts affected with ringworm, or of detached epidermic scales. I am not aware of any observations made on sections of the skin of the affected parts. Yet it must be evident that it is only by examining the whole thickness of the skin that any satisfactory knowledge of the laws which regulate the growth of the parasite can be obtained; and I therefore venture to lay the results of a microscopic examination of the skin in ringworm before the Society.

It is not easy to obtain material for such an investigation from the human subject, but this difficulty is not of much importance as the disease occurs in several of the lower animals.

Cases of direct contagion from the dog or the horse to man are of common occurrence, and Mr. Fleming, the Veterinary Surgeon of the 2nd Life Guards, has communicated to me the details of a case in which a dog having become affected by lying on the cover of a horse which had the disease, communicated it to his master. In this instance the same stock (if I may use the expression) passed from the horse to the dog and the dog to man.

For the sake of convenience I have selected the horse for the study of this question, and I take this opportunity of expressing my sense of indebtedness to my friend Mr. Fleming, for the valuable assistance he has afforded me in

enabling me to familiarise myself with the appearances which the disease presents in this animal, and for giving me portions of skin for examination.

The facts which I have now to communicate are the results of an examination of two patches of ringworm skin from a seven-year-old mare, one of them being very recent, and the other of some duration. As the differences in the development of the characteristic appearances in the two specimens are only of degree, it is unnecessary to insist on them further.

The spores of the *trichophyton tonsurans* are found in and immediately surrounding the hair shaft, and plentifully between the latter and the internal root sheath. In this position they travel downwards from the free surface of the skin along the hair shaft to a considerable depth, but in no instance did I, in my sections, find them reach to the hair papilla. They travel deeper than the level of the sebaceous glands. Not only do they not extend laterally to the external root sheath, but they do not penetrate the internal root sheath, and they are never found in the substance or openings of the sebaceous glands. On the surface of the skin they are only found amongst the most superficial scales of the epidermis. If, therefore, by "crusts" Küchenmeister understood these superficial scales, and by the epidermis the rete mucosum and the deeper strata of the horny layer, my results are in accordance with his. They do not accord with the views of Kaposi.

The significance of these facts will be understood if we consider the relations which the different structures surrounding the hair shaft bear to the epidermis. These relations are acknowledged to be as follows: The hair shaft, with its cuticle, represents a special development of the horny layer, and the external root sheath is nothing more than a continuation downwards of a funnel-shaped projection of the rete mucosum. The layers of epidermic cells, which compose the internal root sheath, represent the cells of the lowermost strata of the horny layer. The parts of the hair with which the spores are found in contact are thus analogous

to the superficial epidermic scales of the horny layer of the epidermis. That is to say, the spores are found amongst lifeless epidermic products, and do not exist amongst those which still retain even the smallest degree of organic life.

The growth downwards of these spores in and around the hair shaft produces directly changes in the shaft itself, and indirectly very striking and important changes in the part of the corium which is near the hair.

At different positions of the hair in the cutis the shaft becomes bent, first at an obtuse, and then nearly at a right angle, and finally it breaks. The broken end of the upper part is then sometimes curled in a semi-circular form, the convex surface downwards, and there is a corresponding bulging of the external root sheath. The lower part of the shaft retains its strait position. These changes may be thus explained. There is a continual growth of the hair from the papilla by which the shaft is pushed upwards towards the surface. When the shaft has become weakened by the disintegrating effects of the growth of the spores, it first bends under the pressure from below, and finally breaks. The lower part of the shaft is then pushed past the detached upper part. The bending of the shaft produces certain mechanical effects on the external root sheath, which bulges and becomes wider.

In one section I found the outer part of it left in its original position in the skin at the level of the bent part of the shaft, whilst the inner part followed the displaced shaft.

In another section a different effect was seen. The internal root sheath had ruptured, and the black pigment of the hair shaft had streamed into the external root sheath, producing at one part decided changes in the cells in a circumscribed part of this sheath. But no spores were found amongst the escaped pigment, or in the affected part of the root sheath.

The fracture of the shaft frequently takes place at the level of the sebaceous glands.

The changes which take place in the skin surrounding the affected hairs are those characteristic of inflammation, from whatever cause it is produced. Microscopically, they consist

in the presence of numbers of colourless blood-corpuscles in and around the walls of the blood-vessels, and although to a less extent, between the bundles of connective tissue. At parts where the changes are advanced, the rete mucosum is found to contain colourless blood-corpuscles between its cells, and in a still more advanced stage, first the mucous layer, and finally the horny layer are completely broken down and all traces of them may have disappeared. In the latter case the free surface is covered with colourless blood-corpuscles (in this situation termed pus cells), and the degree of cell infiltration in the tissues of the cutis immediately under the surface is extreme. The superficial blood-vessels are in this case found distended with blood-corpuscles. But even in such cases not a single spore is found except immediately surrounding the hair shaft, both root sheaths and the connective tissue of the cutis being absolutely free from them.

In some sections oval or pear-shaped cavities were found in the rete mucosum, accurately limited by an even contour, and extending downwards into the upper part of the external root sheath. These cavities were filled with colourless blood-corpuscles, and amongst them a few single detached epidermic cells were found. The cavities were in the strictest sense of the word small abscesses. They contained no spores.

The manner in which these inflammatory effects in the cutis and rete mucosum are produced is hardly susceptible of direct demonstration, but it may be inferred if we take the facts I have adduced in connection with some of the laws which regulate the diseased processes comprised under the term inflammation. The growth of the spores in the hair shaft presupposes a decomposition of organic substances, some of the products of this decomposition being utilised by the growing fungus, others being left unassimilated. If, which is highly probable, some of these products are soluble, they will find their way between the cells of the root sheaths to the substance proper of the cutis. There they act on the tissues as irritants, and, as is always the case in similar circumstances, their action on the walls of the blood-vessels conduces to an escape of colourless corpuscles

from the vessels into the spaces of the surrounding tissue. These escaped, colourless corpuscles, may be so few in number that they are only found scattered singly through the cutis; or the injury to a limited part may be so severe as to cause them to accumulate as the pus cells of small abscesses.

The injury to the coats of the vessels can be recognised in the deepest layers of the corium, and at a considerable distance from the spores. It is made evident by the presence of colourless corpuscles in considerable numbers immediately external to the outer coat of the veins in the lowermost strata of the cutis, and in parts where the cell-infiltration between the bundles is insignificant.

The injury is not always limited to the blood-vessels and connective tissue of the corium. The epidermis itself is sometimes the seat of abscess, or is completely broken down both in its mucous and horny layers.

In addition to the irritation, or, as I believe it to be more accurate to describe the process, the destruction produced by the absorption of soluble substances foreign to the healthy tissue, a certain weight must be assigned to the effects of pressure. When the hair bends, the pressure of the lower part of the shaft is at the point of flexure no longer in a direction parallel to the root sheaths. On the contrary, at this point the root sheaths bulge outwards on one side, causing both pressure and dragging on the connective tissue of the cutis. A certain amount of pressure must also be secondarily induced by the escape of blood serum from the vessels. Although I have no direct evidence of such effusion, we know it must exist, for it is now established that it is the necessary and invariable concomitant of the escape of the colourless corpuscles in all inflammatory processes.

The relative weight that must be assigned to these two causes of inflammation, the existence of which I believe to be matter of legitimate inference, cannot be established with accuracy; but I am disposed to believe that the most important factor is the destructive influence exerted by the absorption of the soluble products that attend the growth

of the parasite in the hair; and for this reason. A great amount of cell-infiltration is often found surrounding hairs in which the spores, although present in considerable number, have produced no distortion.

I will not attempt to discuss all the various phenomena connected with the growth of the ringworm fungus, which are capable of reasonable explanation on the basis of the theory laid down in this paper. That theory is that the spores, whilst only capable of assimilating organic animal substances when these have been severed from the living body, yet exert an injurious influence by the production of hurtful soluble products in a position which facilitates their absorption; and also more indirectly by the results of pressure. The periods of life which are known to favour the occurrence of ringworm, and the conditions of health which are presumed to render it obstinate and its cure difficult, do so, it may be imagined, by combining certain anatomical conditions of the hair follicle, of whose nature we are ignorant, with a languid circulation, which enables the spores to escape contact with the living animal fluids which are fatal to them. Still that is only a subject of conjecture, although very plausible conjecture. It will be more profitable to examine the bearing that the theory has on treatment.

Ringworm of the scalp is treated by two classes of remedies, parasiticides which are also more or less irritants, and irritants pure and simple. It is needless to remark that experience shows that a large proportion of cases are cured by irritants of any kind whatever. Strong acetic acid, blisters, tar, irritating ointments containing mercurial preparations, are examples of such irritants. They have no essential property in common, except that they produce inflammatory action and consequent effusion of serum, and they all cure ringworm. It is, I believe, the inflammation that cures the ringworm. This opinion is strengthened by the fact communicated to me by Mr. Fleming, that in the horse patches of ringworm are cured if they are wounded by a cutting instrument.

The action of parasiticides is mostly, I believe, in the same direction. The parasiticide effect is of course exceedingly valuable, inasmuch as it destroys spores on the free surface, and thus gets rid of a source of danger to unaffected hairs, but a study of the sections described in the first part of this paper has satisfied me that it is very improbable that local applications can reach the spores that are in the deeper parts of the hair.

In some children ringworm may last for years, and may resist frequently provoked inflammation, and the persistent use of parasiticides. Next to carefully performed epilation—a treatment that is in many cases difficult effectually to carry out—the best treatment in such cases is to use an application that will keep up a slight degree of congestion of the affected skin over a considerable period, combined of course with some means of preventing spores taking fresh root on the surface. This principle is well understood by practitioners who have had much experience of these cases. The theory by which it is explained follows naturally from the facts I have adduced. A persistent effusion of serum, sufficient to penetrate the hair sheaths and bathe unceasingly the spores that surround and infiltrate the hairs, acts destructively on the fungus. Between this vegetable parasite, at least, and the living fluids of the body, there is no *modus vivendi*.

The delicate health that is undoubtedly frequently associated with ringworm must, if not caused, be aggravated by it. It is impossible for a chronic inflammatory process to exist without injury being inflicted on the system as a whole, and in this special instance there are causes at work which may be more hurtful than may at first sight appear. We have seen that the vessels are specially attacked, and it is more than probable that the diffusible substances which injure the walls of the blood-vessels pass into the general circulation, and the blood becomes contaminated with noxious matters.

[Some of the portions of skin examined were hardened in alcohol, and some in bichromate of potash, and subsequently

in alcohol. For permanent preparations those hardened by the latter method are best, and double staining with eosine and logwood is very advantageous. When sections from skin hardened in alcohol are first soaked in water, and then macerated in solution of potash, and examined either in the solution or in water, the position of the spores and mycelium and their relation to the structures of the cutis are brought out with great distinctness. Such preparations are useful for controlling preparations hardened in bichromate of potash and preserved in glycerine.]

DESCRIPTION OF PLATE XII.

The Skin in *Tinea Tonsurans*. (Dr. George Thin.)

[Figures 1, 2, 3, 4, and 8, are drawn as seen by a low magnifying power (Hartnack, ocular 3, objective 3). (The existence of spores in the hairs was ascertained by the use of a higher power.) Figures 5, 6, and 7, are drawn as seen by a higher power (Hartnack, ocular 3, objective 8, tube in).]

Fig. 1.—*a*, External root-sheath; *b*, connective tissue of the corium (the dots represent colourless blood-corpuscles); *c*, a portion of the external root-sheath which has become separated by the distortion of the hair; *d*, a sebaceous gland. Its form has been changed by the pressure of the hair.

Fig. 2.—*a*, External root-sheath; *b*, pigment which has escaped into the external root-sheath; *c*, *d*, *f*, sebaceous glands; *e*, a circumscribed patch of cells in the external root-sheath, the cells staining deeply by logwood. Pigment has escaped from the hair shaft into the patch.

Fig. 3.—*a*, The epidermis; *b*, a papilla; *c*, connective tissue of the corium with cell infiltration. (The shaft of the hair is bent.)

Fig. 4.—*a*, Connective tissue of the corium; *b*, a sebaceous gland; *c*, external root-sheath. (The hair-shaft broken.)

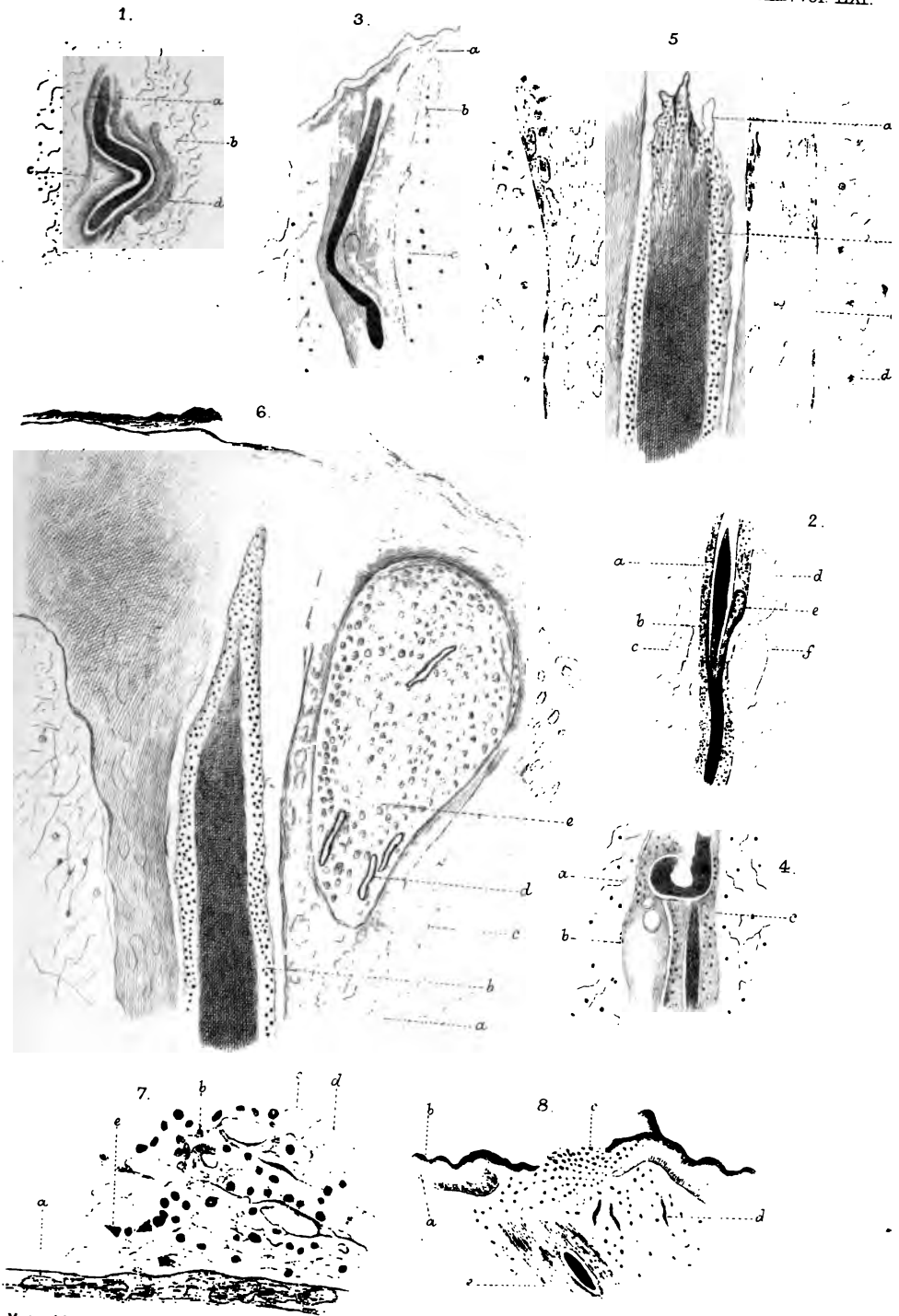
Fig. 5.—*a*, Cells of the internal root-sheath; *b*, spores of the *trichophyton tonsurans* lying between the internal root-sheath and the hair shaft; *c*, the external root-sheath; *d*, connective tissue of the corium.

6.—*a*, External root-sheath; *b*, spores of the *trycophyton*

tonsurans lying between the internal root-sheath and the hair shaft; *c*, connective tissue of the corium; *d*, detached epidermic cell lying in an abscess in the rete mucosum; *e*, cavity in the external root-sheath and rete mucosum filled with colourless blood-corpuscles—(an abscess).

Fig. 7. (To show the manner in which the cell-infiltration in the cutis is produced.) *a*, The external border of the external root-sheath of a hair in which the existence of spores was ascertained; *b*, a blood-vessel. The small round cells in the drawing represent colourless corpuscles, those on the vessel being either in or immediately external to its wall; *c c*, bundles of connective tissue, cut transversely; *e*, a capillary blood-vessel—the vessel was indicated by a line of colourless blood-corpuscles, amongst which a nucleus of the vascular wall could occasionally be distinguished. After a longer course than is shown in the drawing, it approached close to the root-sheath of the hair.

Fig. 8.—*a*, rete mucosum; *b*, horny layer of the epidermis; *c*, mass of colourless blood-corpuscles (pus cells) on an exposed surface from which the epidermis had disappeared; *d*, blood-vessel distended with red blood-corpuscles; *e*, hair cut obliquely. (The hair seen by a higher power to be filled with spores.)



Mintern ich.

Mintern Bro* imp.



ONE HUNDRED CASES
OF
PARACENTESIS OF THE TYMPANIC
MEMBRANE,
WITH THE RESULTS OBTAINED THEREFROM, AND
REMARKS ON THE METHODS OF OPERATION.

BY
W. LAIDLAW PURVES, M.D.,
AURAL SURGEON TO GUY'S HOSPITAL.

(Received January 8th—Read March 26th, 1878.)

THE methods employed have been the knife, the trephine, acids, oils and ointments, Pagenstecher's and other mercurial preparations, the glycerine extract of pancreatine, and the galvano-cautery.

Of these, the knife, Sir Astley Cooper's method, is the most ready and expeditious, but unless combined with some other method, it has been in my hands useless, if a permanent opening is desired. For the purpose of giving exit to fluid, or for making a preliminary perforation, it is to be preferred above all others. A broad needle with cutting edges is a convenient form of instrument for this purpose.

The trephine—Fabrizzi's method—is troublesome in use, and painful to the patient, two objections which are not counterbalanced by any advantages it possesses over other methods.

The same may be said of the use of acids—Simrock's method—but their use has the advantage of not alarming the patient so much as the knife or trephine. The size of the opening can be regulated more exactly by these than by the cautery, but in other respects the cautery is to be preferred. This method also requires, as a rule, more than one application before a perforation is obtained.

By the frequent application of ointments a cataractous drum may be thinned and at last perforated. If the patient can be taught to apply the ointment properly for himself, the combination of this with other methods offers fair prospects of success.

Glycerine, and the glycerine extract of pancreatine, have not been successful by themselves; but this method of digestion, suggested to me by Dr. Lauder Brunton's chapter on digestion, in the 'Handbook for the Physiological Laboratory,' is, I think, worthy of further trial. Other digestive secretions, such as pepsine, might be used, either alone or in combination with the cautery or knife.

The galvano-cautery—Voltolini's method—I prefer to all others. The following is the procedure which I have found best:—Having ascertained by perosseal audition that the acoustic is fairly healthy, the hearing power is determined by the watch. A preliminary puncture is then made by means of a broad needle, and while the opening gapes, and is still unaffected by the congestion arising from the irritation, the hearing power is again ascertained. If it has improved, the galvano cautery, if ready, may be at once applied to the spot, and the opening made as desired. If the operation is not to be completed at that sitting, cotton wadding is placed in the meatus, and the patient is directed to return after the lapse of a week or a fortnight. Should the hearing not be improved by the preliminary puncture, wool is placed in the ear, and nothing further is done.

The opening having been made by the cautery, a pad of wadding smeared with mercurial or simple ointment, or the glycerine extract of pancreatine, is passed into the opening and pressed against the drum, so as to expand it as much as

possible, and left there for two days. This causes the wound to gape, and at the same time acts, I believe, prejudicially on the nutrition of the drum. The wadding is removed every second day, the parts washed with warm water and carbolic acid solution, the drum drawn outwards by means of the vacuum speculum, for the purpose of preventing any adhesions forming, and the wadding again applied. This has been hitherto the most successful method which I have tried, but this success is not sufficiently satisfactory.

If a method of causing the drum to bulge externally can be arranged, the danger of adhesions which the above method entails would be avoided.

When a large cicatrix of an old perforation is present, I choose that spot in preference, hoping, as proposed by Weber, Liel and myself (*vide* 'Guy's Hospital Reports,' 1875), that the nutritive processes caused by the irritation will be less exalted in that spot, and I have found that in cases having such cicatrices the success has been greatest. Frequent repetition of the perforation in the same spot has a similar effect, and I have seen benefit in some cases by the thinning of the drum, which is gradually established at the spot, though no perforation remain.

The cautery employed may be modified in shape according to the action desired. If a permanent aperture is required, a thin wire, with an expansion at the active end, is convenient. If it is employed merely as a tightener in a relaxed drum, a thicker wire, or several points, will be found useful.

The cases in which I have tried the operation have been : (1) thickening of the drum, (2) relaxation of the membrane, (3) tinnitus, (4) accumulations in the cavity, (5) adhesions in the cavity, and (6) in chronic cases in which other remedies had failed as a dernier resort. It is also used in cases of impassable stricture of the Eustachian tube, and I would certainly employ it in such, if a case occurred in my own practice.

Of 83 individuals operated upon, 46 were improved temporarily or permanently. If the causes, however, which induced operation are considered, the benefit received by certain

classes of cases was much greater than appears when looking at the general success. Of 8 cases in which paracentesis was performed on account of tinnitus, only 2 received benefit from the operation, 5 were not improved, and in 1 the result is not given. Of 6 cases of nervous affection, in which the operation was exploratory, or as a dernier resort, not one was improved. In 2 cases, the operation was merely a preliminary step to further action on adhesions in the cavity, in one of which the operation was successful. Of 39 cases of cataractous drums, 23 were benefited, 12 were not, 3 were doubtfully improved, and in 1 the result is not given. In 7 cases of relaxed membranes, paracentesis improved 5, the remaining 2 were not improved. In 21 cases in which the perforation was made for the removal of fluids or solids from the cavity, 15 were benefited, 5 were not, and in 1 the result is not given.

The operation was performed 104 times. In some cases the benefit was so great that it was repeated several times at the request of the patient. This occurred especially among those with cataractous drums. In no case have I seen any alarming results follow the operation.

In one case, Case 19, all the methods mentioned, except that of digestion, were tried. This patient has been deaf since childhood, and had two dense immovable drums. The hearing power on the left side was $\frac{0}{1x}$ and on the right $\frac{1}{1x}$. Choosing the worse ear, the slit made by the knife improved the hearing at once, and on making a large perforation by means of sulphuric acid, it improved to $\frac{18}{1x}$, and while lying in bed the patient could hear the tick of a clock hanging on the wall, which he said he had not been able to hear before. In ten weeks he returned with the opening closed, and asked me to repeat the operation. The opening was again made with acid, and he was advised to paint the edges of the perforation with glycerine and carbolic acid by means of a small camel-hair brush. Eight months afterwards he returned to have the opening again made. This was done by the cautery, which was used at the same spot in hopes that the cicatricial tissue would be less likely to have such a renovating power

as the untouched membrane. This opening, however, closed, and the cautery was again applied at the same spot, and the perforation was present when I last heard of the patient some two weeks after the operation.

In case 15, the improvement received by the first operation was sufficient to cause the patient to come to town to have it repeated. Choosing the cicatrix of the old opening, acid was used, and after making a large perforation, wadding smeared with simple ointment was pressed against the membrane. This he renewed for himself daily. The perforation was present when the patient was last heard of, seven weeks after the operation.

Case 23 had been operated upon by Mr. Hinton, 10 years before, and the patient had received so much benefit from that paracentesis, that he was desirous that it should be repeated. The perforation was made this time by the cautery, and the improvement was again considerable.

The choice of the part of the membrane for operation is, I think, somewhat important. If the perforation is made for the purpose of removal of fluids from the cavity, it ought to be made in the portion which bulges most. If a relaxed membrane is the cause of the operation, the portion showing the greatest want of tension is chosen, and one or more slits made according to the degree of relaxation. If a permanent opening is desired, I prefer a point posterior to the manubrium, and midway between it and the periphery, because this point is furthest from the blood supply, and it allows the passage of waves of sound in a moderately direct line to the secondary membrane.

No.	Initials.	Age.	Sex.	Reasons for operating.	Method.	Position of perforation.	Result to hearing power at time of operation.	Time the perforation remained open.	Remarks.
1	H. L.	...	R.	<i>Thickened membrana tympani</i>	Knife	Post. inf. quadrant	Improved		
2	A. B.	22 R.	"	"	Trephine and knife	"	O $\frac{5}{1X}$ to $\frac{1}{1X}$...	Patient returned in 6 weeks saying that as long as he kept cotton-wadding in the ear the perforation kept open, but now closed. Again perforated, and this closed in thirty hours.
3	J. W.	60 R.	"	"	Trephine	"	C $\frac{1}{1X}$ to $\frac{1}{XL}$		
4	J. W.	60 L.	"	"	"	"	C $\frac{1}{1X}$ to $\frac{1}{XL}$	3 months	
5	W. M.	23 R.	"	"	"	"	Improved	6 months	
6	W. M.	23 L.	"	"	"	"	"	30 hours	Made a second paracentesis on same spot.
7	J. F.	45 R.	"	"	"	"	"		
8	T. H.	64 L.	"	"	"	"	Improved to conversation, not to watch		
9	S. K.	67 R.	"	"	"	"	Much improved.	8 days	Again paracentesed at patient's desire.
10	W. E.	51 L.	"	"	"	"	Improved	...	Repeated in 7 days.
11	M. W.	56 L.	"	"	Knife	"	No improvement	2 days	
12	L. R.	50 R.	"	"	"	"	Improved		
13	H. G.	32 R.	"	"	Trephine	"	No improvement		
14	E. W.	25 L.	"	"	"	"	"	...	Perosseal audition imperfect.
15	J. C.	38 L.	"	"	Knife and sulphuric acid	"	Improved	...	This case was left alone till the cicatrix of the preliminary incision had formed, and then sulphuric acid was employed, and after the opening had been made, a plug of oiled wadding was pressed upon the

[illegible]

No.	Initials.	Age	Reasons for operating.	Method.	Position of perforation.	Result to hearing power at time of operation.	Time the perforation remained open.	Remarks.
23	A. H.	30 R.	<i>Thickened membrana tympani</i>	Knife and cautery and wadding	Post. inf. quadrant	C $\frac{4}{LX}$ to $\frac{4}{LX}$	Open when last seen	This patient had been operated on in the same ear by Mr. Hinton 8 years before, and then heard better after operation. The old cicatrix was still to be seen.
24	S. K.	28 L.	"	Knife	"	No improvement	...	The membrana tympani was very concave externally, and a case for tenotomy.
25	W. S.	40 R.	"	" and cautery	"	$\frac{1}{LX}$ to $\frac{3}{LX}$	Open when last seen	
26	E. G.	... L.	"	"	"	Improved	"	
27	W. Y.	18 L.	"	Knife	"	No improvement	3 weeks after operation	
28	G. B.	52 R.	"	"	"	"	...	+ 2 concave membrana tympani.
29	W. B.	29 R.	"	"	"	"	...	Affection of acoustic.
30	R. M. F.	19 L.	"	" and cautery	"	Improved	Open 12 days after; not seen since	Tinnitus not improved.
31	G. T.	27 R.	"	Knife	"	No improvement	"	Affection of acoustic.
32	A. H.	33 L.	"	"	"	"	...	" (specific).
33	A. H.	33 R.	"	"	"	Doubtful improvement	3 days	Again perforated, and again closed in 72 hours. The improvement being slight it was not repeated.
34	E. B.	37 R.	"	"	"	Result not given	...	Wadding saturated with glycerine pressed into opening.
35	E. B.	36 L.	"	" and cautery	"	Improved	"	
36	M. S.	23 L.	"	"	"	"	"	
37	R. P.	54 R.	"	"	"	"	"	
38	M. A. H.	38 L.	"	Knife	"	"	"	
39	J. G.	28 L.	"	Trephine	"	Improved.	"	

	tympani	phuric acid	"	"	"	"	"	"	"
42	F. M. 18 R.	Relaxed cicatrix, the rest of membrana tympani being dense	Knife	"	"	"	"	"	"
43	R. F. 35 ...	Relaxed membrana tympani at old cicatrix, which flapped extensively	"	"	"	"	"	"	6 months
44	A. S. R. 28 ...	Relaxed membrana tympani	"	"	"	"	"	"	"
44A	A. P. 16 L.	Relaxed membrana tympani, the greater part of the membrane having been lost at one time	Cautery	"	"	"	"	"	"
45	E. P. 18 L.	Membrana tympani drawn in and atrophied	Knife	"	"	"	"	"	"
46	A. G. B. 37 R.	<i>Tympanus</i>	"	"	"	"	"	"	36 hours.
47	A. G. B. 37 L.	"	"	"	"	"	"	"	36 hours.
48	A. C. 59 ...	"	"	"	"	"	"	"	"
49	E. L. 31 ...	"	"	"	"	"	"	"	"
50	C. I. 45 ...	"	Knife and cautery	"	"	"	"	"	"
51	L. C. 36 L.	"	Knife	"	"	"	"	"	"
52	W. B. 47 R.	"	"	"	"	"	"	"	"

phuric acid for the purpose of heightening the effect of the paracentesis.

The whole membrana tympani seemed to be formed of two large cicatrices.

Healed by a very thin goldbeater-skin cicatrix, which was easily broken down by probe a month afterwards.

No.	Initials.	Age	Reasons for operating.	Method.	Position of perforation.	Result to hearing power at time of operation.	Time the perforation remained open.	Remarks.
52A	E. L.	24 L.	<i>Tinnitus</i>	Knife	Post. inf. quadrant	No improvement	...	Patient had a brother who died of "tumour of the head." This patient came on account of the deafness and fits, which occurred daily before the operation. The mass of the tumour was removed at two sittings. He had no fits after the operation. Seen a week after. Affection of the acoustic.
53	B. B.	20 L.	Removal of substances from cavity. Sebaceous looking matter (molluscous tumour)	Knife, suction by piston	Both sides of manubrium, large incisions	Improved	...	
54	B. P.	33 ...	Fluid which came after sudden sickness	Knife and vacuum speculum	Lower part	Hearing not improved but feels lighter & easier	...	
55	W. W.	Mucus	Knife and suction	"	Improved	...	The patient said this ear had been deaf all his life. In coming out through the slit the mass, though under pressure, was not broken, and when the whole mass fell on to the floor of the meatus it formed a mass of nummular shape. This was a much thinner fluid, seemingly the result of acute inflammation.
56	B. P.	38 ...	Serous-like fluid	"	"	"	...	
57	M. B.	50 ...	Mucus	"	"	0 " $\frac{1}{2}$...	
58	C. E.	52 L.	Thick jelly-like pinkish mass	"	"	LX to LX	...	
59	C. E.	52 R.	Serous-like fluid	"	"	0 $\frac{2}{LX}$ to LX	...	Tinnitus removed almost immediately.
60	F. F.	13 L.	Mucus	"	"	Not given.	...	
61	C. G.	63 R.	"	"	"	Heard at once as well as he had ever heard	...	
62	E. L.	24 R.	" and tinnitus	"	"	$\frac{6}{LX}$...	

No.	Initials.	Age.	Sex.	Reasons for operating.	Method.	Position of perforation.	Result to hearing power at time of operation.	Time the perforation remained open.	Remarks.
75	A. S.	23	R.	To remove adhesions	Knife and vacuum speculum and Politzer	Throughhold	No improvement		
76	J. S.	60	L.	Menière's disease	Knife	Post. inf. quadrant	"		
77	G. C.	27	R.	"	"	"	"		
78	C. P.	20	R.	Acoustic affection	"	"	"		
79	E. J.	24	R.	"	"	"	"		
80	L. P.	49	R.	"	"	"	"		
81	J. W.	45	.	"	"	"	"		

ON SOME POINTS
IN THE
MINUTE ANATOMY OF THE KIDNEY,
AND
THEIR RELATION TO THE PATHOLOGICAL
PHENOMENON OF TUBULAR CASTS.

BY
REGINALD SOUTHEY, M.D.,
PHYSICIAN TO, AND LECTURER ON FORENSIC MEDICINE AT,
ST. BARTHOLOMEW'S HOSPITAL.

(Received January 8th—Read April 9th, 1878.)

SOME few years ago a fresh interest was awakened in the kidney by the announcement of the discovery of two sets of tubes in those bundles of straight tubes which compose the substance of the medullary rays of the kidney.

Observers were agreed in acknowledging the existence of these two kinds of tubes, termed from their respective courses, ascending and down-looping tubes, but the connection of the one set with the other remained a disputed matter, and Henle, to whose original investigations their discovery was due, believed them to form separate systems of channels, the one occupied, like closed gland tubes, in secreting the materials, which, after transudation through two sets of tube walls, the other set collected and discharged.

My own observations, although made many years since,

inclined me to believe that the tubes formed one continuous system, and this belief was based upon examination of fragments of urine tubes, which although obviously forming one continuous channel, differed very distinctly in diameter, calibre, and characters in different parts.

My observations were made upon the kidney of the ox, the sheep, the pig, the rabbit, and that of man; and some microscopical preparations of my own are on the table, to substantiate my views, and also to render the scheme of the real anatomical arrangement of the urine tubes easily intelligible to any one.

Mode of Commencement and Course of:—The *Tubuli Uriniferi*. To commence from below upwards. The papillary ends of the medullary cones are studded with from ten to thirty oval orifices, these open into short trunks, which, instead of mounting perpendicularly upwards, run as offshoots, or runners, at very obtuse angles from the original trunks. The short trunks, and their first angular bends, possess no *membrana propria*; the channels are simply grooved passages in the fibrous connective tissue forming the matrix of the papillæ; they measure from 0.198 to 0.099 mm. in diameter.

This arrangement offers mechanical resistance to regurgitation of fluids accumulated in the renal pelvis. The angular mains quickly divide into three or four mounting stems, and these shortly split up again into the ascending or collecting tubes proper; tubes of the third order as described by the minute anatomist.

These ascending tubes are the first which can be distinctly made out to possess a *membrana propria*; they are lined with a low sessile columnar epithelium, consisting of pale cubical cells, streaked with indistinct markings, and possessed of nearly central nuclei. The epithelium does not nearly fill up the bore of the tubes, which therefore exhibit large lumina or bores.

These ascending or collecting tubes of the third order are large tubes, and measure about 0.051 to 0.0501 mm. in diameter, at a point about 2 lines off the apex of a papilla.

They are well shown in all works which describe the minute anatomy of the kidney, and form the principal bulk of the medullary rays.

At a level some 2 lines higher as they mount towards the cortex, or, at what is called by the anatomists the cortical margin of the medullary ray, these collecting tubes, hitherto all bundled together, begin to split into branches of nearly equal size, 0.018 to 0.02, and they continue running up in bundles of three or five together; the space between the bundles being occupied by tubes presenting different characters, the down-looping tubes of Henle, which are transparent, and at parts much narrower tubes, measuring about one third their diameter, only 0.0099 mm. They are well shown in Fig. 1, made from a microscopic section cut longitudinally at the margino-medullary level.

I wish to direct attention next to the number of the down-looping tubes (well shown in Fig. 2), this drawing is made from a carmine stained specimen of great beauty. The excretory tubes, were so far washed clean of their epithelium that the down-loopers, which in this part possess no distinct epithelial linings, become apparent, and the nuclei of their endothelium afterwards stained with carmine, brought their number and arrangement into distinct relief, as also their far greater predominance at the upper or high levels of the medullary rays or cortical side of the pyramids. (It was the difficulty of throwing coloured injections so far as these down-looping tubes, which militated, in the first instance, against their discovery, and the likeness of their epithelial linings to that of blood-vessels which prevented their being recognised for what they really are, namely, continuations of urine tubes.)

Tracing the collecting tubes in their upward course, after they have entered the so-called cortical portion, in which bundles of straight and tortuous tubes alternate with each other, the collecting tubes can be seen to turn over at various different levels into the tortuous tube columns, and to become tortuous tubes, suffering two substantial changes at the same time. 1st. An alteration in calibre, for they

attain suddenly the full size of tortuous tubes proper, averaging 0.0201 in diameter; and, 2ndly, a marked difference in the characters of their cellular contents, which are far less distinctly differentiated, the individual cells being larger, more irregular shaped, and appearing nearly to fill the urine tube with finely granular cubical cells; these cubical gland-cells are admirably shown in Figs 3 and 4.

No drawing from a microscopic section will take in the whole course of an ascending collecting tube, through its first sudden tortuous enlargement, and its final gradual tapering with a down-looping prolongation, but several sections and preparations in my possession, of which I invite inspection, enable any one to follow single tubes up and down, passing into and becoming down-loopers. Figs. 3 and 4 are drawn from sections prepared by Mr. Pye, and most faithfully represent the facts of the case.

It is, however, only for a very short space that these now tortuous tubes maintain their tortuous characters, for they turn back into the straight tube bundles, and run down, as previously noticed, between the collecting tubes, as the fine transparent down-loopers, which are seen to be lined with flattened pavement epithelium, surmounted at pretty regular intervals by slightly projecting nuclei, and, indeed, resembling capillaries very closely both in size and endothelium.

The passage of such a tortuous into a straight down-looping tube is shown in Figs 3 and 4. Fig. 4 delineating the excretory or collecting tube end, and Fig. 3 the secretory or glandular tube end. As these narrow tubes retrace their passage upwards, they increase, not suddenly but gradually, in size, like carrot roots, presenting at first a distinctly imbricated columnar epithelium, then curling over again at different levels into the tortuous tube columns, and finally becoming lost as tortuous tubes proper, being filled throughout their interiors, as these always appear to be, with granular, glandular cells; each tube terminates, after many turns and windings, by a somewhat sudden, narrow neck and flask-like dilatation, which embraces a Malpighian tuft.

The tortuous tubes measure in diameter upon an average 0.0201 mm., but they vary, not inconsiderably, in size at their turns, as may be observed in fragments of them prepared by cooking in hydrochloric acid. Their main feature is the thickness and apparent solidity of their walls, and its dimly nebular aspect.

Put up as microscopical specimens it may be surmised that we see them under conditions very different to those which they present as fresh objects, derived from the perfectly fresh kidney; no bore or patent passage is then apparent in them, except when an injection fluid forced up into them discovers itself by irregular stellate figurings, marking the chinks between opposed walls, or separations between masses of granular protoplasm, in which apparently free nuclei lie imbedded.

We read in Stricker, p. 93, "That the basement, or limiting membrane, of these tubes is as clear as glass, and the degree in which the epithelial mass fills the bore of the canal, depends upon the state of the kidney at the time of death, and its mode of treatment afterwards." With this description my own original observations accord entirely.

If this account of the anatomical course of the tubuli uriniferi be accepted as correct, certain obvious inferences follow. From the size of the tubal canal at different parts of its course: nothing larger than nuclei, blood-cells, or leucocytes, could pass down from the tortuous or glandular part of the tubuli through the down-looping prolongations, so as to reach the straight collecting tubes. A cast shed from a tortuous tube, could only under circumstances of long-continued pressure *à tergo*, and extreme dilatation of the down-looping canals, reach the collecting or excretory uriniferous channels.

As a matter of fact there is no such cell lining attached to the basement walls of the upper portion, the secreting portion, of the tortuous tubes, as belongs to the lower excretory or collecting canals. By examining the Figs. 3 and 4, it must be obvious that, unless the drawing is out of scale, a gland cell in its ordinary shape, as seen in normal

states of the glandular portions of tortuous tubes, could not squeeze itself through the channel of a down-looper. When, therefore, a cast is assumed to be derived from the profounder tissues of the kidney, and to have a relatively graver importance attaching to it, because cells are seen in its interior, an error is committed, based on ignorance of the minute anatomy of the organ.

Finely granular plasma containing fatty dottings, oil globules, and nuclei, are the derivatives and detritus which can fairly be referred to the upper portions of the tortuous tubes; since these can be often seen lodged *in situ* in their interiors in sections of kidney advanced in acute or chronic degeneration, and can without much doubt be gradually extruded so as to reach down into the lower channels, and washed from out of these; but the large gland cells massed together as desquamative casts, could not pass the narrow passage, and the synonym, desquamative nephritis, is a term which I must enter my humble protest against.

Anything resembling the epithelial lining of the large, straight, or collecting tubes shed in their entirety, akin to the desquamation of the cuticle after scarlet fever, it has never been my lot to witness, nor do the pathological appearances of the kidneys, in those who die of this acute nephritis, lend any confirmation to the hypothesis that the excretory channels are then denuded of epithelial contents. They are not found empty or collapsed, but largely swollen and blocked up. The straight collecting tubes, however, are seen oftentimes in chronic nephritis plugged or blocked with materials, which entirely correspond to those discovered in the sediment of the urine during life, and which are derived from the upper secreting gland structures.

The actual cubical columnar epithelium cells, which line the tubuli in various parts, I never yet saw in recognisable character in any renal cast; the formed objects which pass for renal cells with the ignorant, are red or white blood-cells or escaped nuclei.

Desquamative nephritis, in the sense of any desquamation of the epithelium, which, in healthy kidneys, certainly lines

the collecting and straight tubes, as a process at all, akin to the peeling of the skin which succeeds the slight serous effusion beneath the horny layer of the epidermis in the scarlatinal efflorescence, does not exist.

Our present better knowledge of the minute anatomy of the kidney, enables us to offer a less crude pathological solution of the mode of cast formations than was offered heretofore; when the assumption of some writers was, that renal inflammation led first to the shedding of the tubal epithelium, and secondly to the transudation of blood serum as a consequence of this denudation of the tubes.

Axel Key, the Swedish Professor, whose opinion upon casts and their formation is entitled to some weight, for his patient investigations into the anatomy and pathology of the kidney are known throughout Europe, allows that certain casts originate in the degeneration of renal epithelium; these elements melting down into masses more or less homogeneous or granular, but preserving no outline of their original cell forms. Certain other casts, he believes, are perverted or abnormal secretions derived from disordered functioning of the secreting cells. To these we may fairly add a third form, which consists of blood fibrine entangling more or less blood elements. For clinical purposes I should distinguish these three varieties only, calling them granular, waxy, and blood plasma casts respectively.

Casts occur in every form of renal disease in which albumen appears in the urine, although their quantity is no indicant either of the amount of albumen, or of the amount of renal degeneration.

The first value attached to the presence of casts, and to the characters of casts, by those who busied themselves most with renal disease, was very high, too high perhaps. It was supposed that being derived from the minute kidney structures, they would tell an accurate tale of the processes of disease taking place in those structures, according to their numbers and other special features.

Before even the minute anatomy of the organ, or the intricate course of the tubuli uriniferi was established, it

was pretty generally accepted that the larger casts, and those which presented most distinct cellular elements in their interiors, were derived direct from the large tortuous tubules. Still, experience taught that these large casts, with leucocytes in them, were of not very serious omen. They were furnished in greatest abundance in the re-establishment of the urinary secretion after temporary suppression in acute nephritis.

In point of fact these large casts correspond in size entirely with the diameter of the excretory canals and straight collecting tubes measuring about 0.05 mm. in diameter, and are no doubt formed in and derived wholly from them. Furthermore, they may be seen *in situ* in many pathological specimens of which I exhibit examples. The observation has been so repeatedly made that illustrative drawings are deemed unnecessary.

The next fact, clinically well ascertained, was the grave import of granular, waxy, and fat-dotted casts, as signifying changes of a chronic nature in the secretory portions of the organ. These casts too, though usually more fragmentary than those previously described, still ranged in size only from 0.01 to 0.05 mm. in diameter. Yet they were supposed to be derived from the tortuous tubes, and, indirectly, no doubt they were. But it is a different thing to be derived from a part, and to be moulded in that part. The cells which block up a tortuous tube could no more pass through a down-looper without altering their shape, than a camel could squeeze itself through the eye of a needle.

Casts are very properly thus entitled, not because the materials which compose them are necessarily cast off from some portion of the urine tubes, but because they are cast in the moulds of tubes, and form solid cylinders. One of the conditions required for their formation is the transudation of the blood plasma into the tubuli, a second is an altered state of nutrition in the renal epithelium, a third the retention of this secretion, thus abnormal, for a certain period, how long we do not know, *in situ*, in one of the collecting tube channels.

The largest casts are those found in the urine after the

suppression which succeeds the algide stage of cholera. These casts are most likely moulded in the large gathering mains near the papillary orifices.

The medium sized casts, although varying much in colour and other characters, according to the nature and duration of the renal disease, in which they are furnished, are probably all moulded in the straight collecting tubes or ascending branches of the third order.

The smallest, finest, most wavy and hyaline, so-called fibrinous casts, are probably chiefly moulded in Henle's down-looping canals.

The formation and excretion of casts are by no means simultaneous events. The more opaque, the more granular, the more dotted with delicate fat globules, they are, the longer probably has been their sojourn in the spot where they were moulded, and the greater their saturation with urinary salts; whereas, contrariwise, the finer and sharper their outline, as well as that of the cell forms entangled in them, and the more transparent their bodies, the more likely are they to have been quickly swept out of the passages in which they were formed.

The gravest forms of renal degeneration are only now and then attended by the extrusion of casts. The pale urine of low specific gravity, and very varying albumen contents, which betokens with such certainty the small cirrhotic kidney, furnishes us with few, and seldom very conclusive, forms of casts. The study of the minute anatomy of the kidneys forbids us now to expect that casts could reflect the amount of degeneration which has taken place in the upper portions or tortuous prolongations of the renal tubes, although indicating, as they certainly do, its nature. Their presence (the presence of casts) in the urine sediment only shows that the entire system of excretory tube channels is not flushed down by urinary fluid with the regularity that belongs to the secreting functions of healthy organs; and that an abnormal secretion, abiding for a longer time than it ought to, in channels, where certain conditions of health are maintained, has gelatinized within these canals and blo-

Microscopic specimens in my possession give examples of granular plugging in the collecting tubes, and huge dilatation of the tortuous tubes above them, a very common event in chronic parenchymatous nephritis. The kidney some were taken from belonged to a young woman who died of this form of renal disease in the stage of advanced secondary contraction. I had carefully watched her case over a period of five years, during which she was repeatedly under my care in St. Bartholomew's Hospital.

Lastly, I will call attention to a fact of considerable interest, and which I possess specimens to illustrate, namely, the manner in which Henle's down-looping tubes, or what I shall now call communicating loops, resist fatty degeneration, and although small themselves in calibre seldom become plugged up with epithelial débris or proper fatty degeneration. They lie, as is well known, in the vascular area surrounded by the vasa recta, their nutrition is therefore provided for amply, although the tortuous tubes above them have been starved of their nutritional supplies, through parenchymatous swelling or degeneration of the Malpighian bodies. Such pluggings as we occasionally do see *in situ*, in the down-looping tubes, are generally fibrinous blood casts, and strictly of a nature which the situation of the tubes themselves would render most probable.

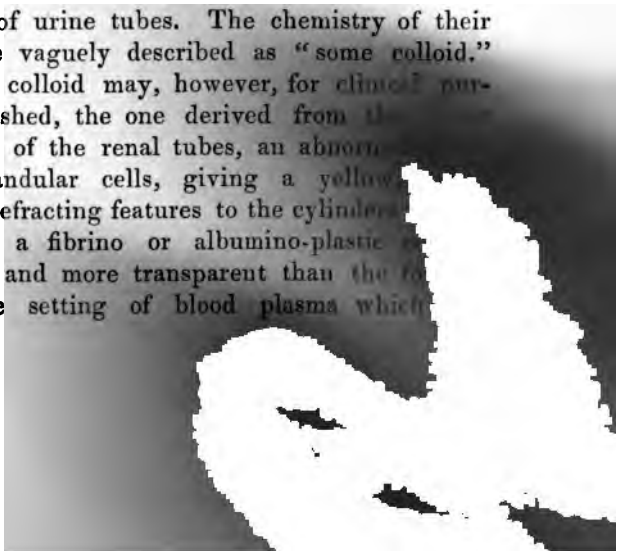
I must ask pardon, however, for having trespassed thus far upon the patience of the Society, and occupied its time so long with a matter of microscopical anatomy so minute. My excuse is, that the pathology of renal disease is, although much written about, and fairly well understood in its later and advanced stages, very imperfectly known, and extremely difficult to study *ab initio*.

The circulation of the blood through the kidney is a very complex matter. The intimate relation of the vasa recta or tertiary capillarisation, to Henle's looping tubes is a subject which has not received the attention which it appears to me to deserve. These looping tubes, so far as I have been able to prosecute the investigation, exist only in those classes of the animal kingdom which excrete urinary water,

their total absence, as well as that of the vasa recta in the kidneys of serpents, lends confirmation to an opinion expressed and published by me so long ago as 1865, in an article communicated to the first volume of 'St. Bartholomew's Hospital Reports,' that the down-looping tubes and vasa recta probably form the apparatus which is most instrumental in the secretion of urinary water.

If the description of the minute anatomy which I have detailed here be correct, and it is, I believe, now pretty generally accepted. It will be obvious that midway canals of smallest calibre are interposed between the secretory or tortuous, and the excretory or straight tubes. If casts or plugs collect in the tortuous tubes, their passage onwards and outwards must be slow and laborious, and to be effected only through some gradual dilatation of the narrow looping passages. Further, such casts by distending the down-looping tubes upon their passage outwards must seriously disturb or obstruct the circulation through the vasa recta.

It is only fragmentary debris and fatty globules, however, which I believe travel down from the tortuous into the excretory tubes; and I may add that even in advanced renal disease, it is remarkable how free from plugging and degeneration these down-loopers usually remain, a circumstance which could scarcely happen if much solid material ever passed through them, or except a strong current of fluid set through the walls of the capillaries by exosmosis towards the down-loopers and swept through their canals. All casts found in urine sediments derive their form from the excretory system of urine tubes. The chemistry of their substance may be vaguely described as "some colloid." Two qualities of colloid may, however, for clinical purposes be distinguished, the one derived from the glandular portions of the renal tubes, an abnormal secretion from its glandular cells, giving a yellowish look, and highly refracting features to the cylinders of it; the other a fibrino or albumino-plastic secretion, essentially whiter and more transparent than the first, derived from the setting of blood plasma which



transuded into the tubuli from the blood-vessels at a lower part of their course.

Several sub-forms or varieties of casts may be further differentiated, because different clinical significance or importance attaches to them. The more distinctly formed cell elements I perceive entangled in cast moulds, the more certainly I infer congestive blood pressure in that portion of the kidney structure, which the ground colour of the cast points out as its probable source. I cannot pretend to be able to state that a particular cell form seen in a cast has been derived from a tortuous or an excretory tube. The cell forms I can recognise are always leucocytes, white or red blood-cells. Hyaline cylinder casts, from their form and size, are pretty certainly derived direct from the down-looping tubes, and appoint obstructed circulation through the Malpighian bodies, and tortuous tubes with blood flux through the vasa recta. Lastly, the more granular a cast is, the longer generally may it be assumed to have resided in its matrix mould, and the more dotted with fat globules casts are, the more certainly do they appoint permanent obstruction to the circulation through those portions of the kidney from which the colloids which form them were derived, and permanent damage, not to the tubes only, but to the capillaries and interstitial structures of the kidney.

It is not, I believe, until some obstruction to the circulation through considerable areas exists, or until this obstruction has been maintained for some little time, that the necrosis of elements implied by fatty degeneration can take place.

To estimate the nature and extent of renal disease by the form and aspect of one or two tube casts, would be to guess in a very blind haphazard manner. Fatty casts are always ill-omened things to find; but even here the room for error in those who make their inferences from casts alone is not inconsiderable. Under such circumstances the only safe guide to a prognosis is to ascertain the functioning capacity of the remaining renal structures by estimating the amount of urea excreted per diem, and the strain of blood pressure

thrown upon the still permeable blood channels, by the diurnal amount of albumen that is being lost. Experience teaches me that it is not so much the drain of albumen which wears out the machine, as it is the blood strain, which that amount of albumen signifies, and which by induced secondary interstitial changes, slowly but surely abrogates the renal functions, and brings about the complex of secondary phenomena attendant upon deficient urinary depuration.

DESCRIPTION OF PLATE XIII.

Minute Anatomy of the Kidney. (Dr. Reg. Southey.)

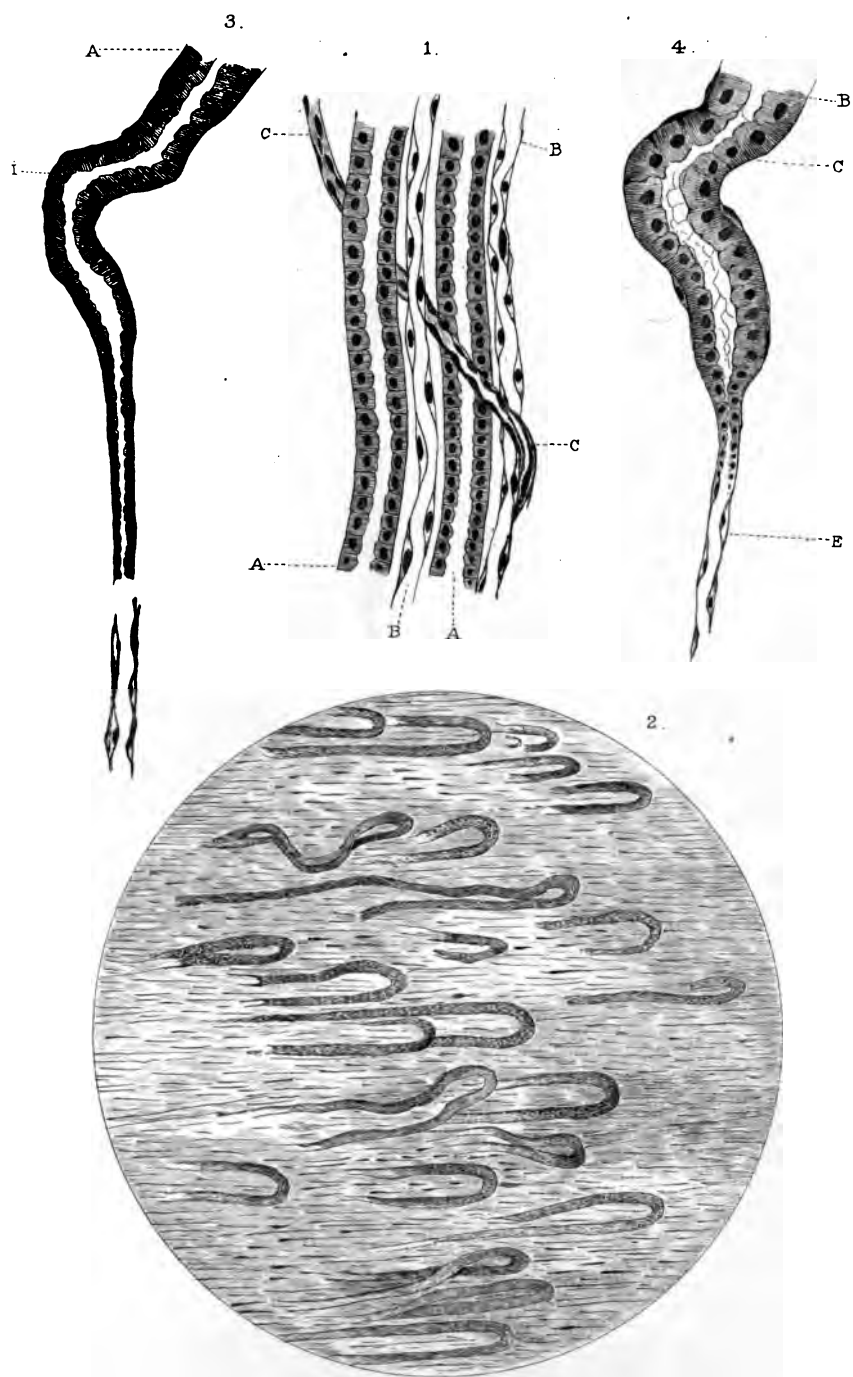
Fig. 1. Drawn from a microscopic section. $\times 100$ diameters.

- A.** Ascending collecting urine tubes.
- B.** Henle's down-looping tubes.
- C.** Capillary blood-vessels.

Fig. 2. Microscopical specimen shown with a low power, illustrating the number and arrangement of the down-looping tubes.

Fig. 3 A. Tortuous tube derived from a Malpighian body, tapering to join the ascending loop. *i.* Imbricated columnar epithelium.

Fig. 4 B. Tortuous tube derived from the tortuous portion of a collecting tube, suddenly narrowing into a down-looper. *C.* Cubical gland cells, with nuclei. *E.* Flattened elongated cells, with projecting nuclei of down loopers.





ON
PARALYSIS OF THE INTERNAL MUSCLES
OF THE EYE

(OPHTHALMOPLEGIA INTERNA).

A GROUP OF SYMPTOMS WHICH PROBABLY INDICATES
DISEASE OF THE LENTICULAR GANGLION.

BY
JONATHAN HUTCHINSON, F.R.C.S.,
SENIOR SURGEON TO THE LONDON HOSPITAL; SURGEON TO THE MOORFIELDS
OPHTHALMIC HOSPITAL; AND TO THE HOSPITAL FOR SKIN DISEASES.

(Received January 8th.—Read April 9th, 1878.)

I AM not aware that any observer has as yet associated any group of symptoms with disease of the lenticular ganglion. Nor am I in a position to prove by an appeal to dissections that any group of symptoms can beyond doubt be so associated. The object of the present paper is, however, to point out that a certain combination of signs would be likely to follow destructive disease of that body; and secondly, to show that we do meet with cases in which precisely this combination is present in the most definite and remarkable manner. All the patients in whom I have seen it are as yet living, and I have therefore had no opportunity for post-mortem dissection.

The lenticular ganglion receives branches from three sources, two of them motor, and one sensory; the third nerve, the vaso-motor, and the fifth. The distribution of the sensory filaments proceeding from the ganglion cannot easily be identified in bedside observation, since the front of the eyeball receives nerve-supply from the other branches of the fifth, and it may be conjectured that no appreciable anæsthesia would result from destruction of the small filaments which pass into the ganglion. The motor roots, however, supply important structures, the iris and ciliary muscle, paralysis of which can easily be appreciated.

The symptoms which *à priori* would be expected to result from destruction of the lenticular ganglion are iridoplegia or paralysis of the iris, both as to its circular and radiating fibres, and cyclo-plegia or paralysis of the ciliary muscle. My suggestion is that when this triad of symptoms is present—inability to use the ciliary muscle, paralysis of the dilating fibres of the iris, and paralysis of the contracting fibres of the iris,—without any other form of orbital paralysis, the seat of disease can be in no other structure than the ganglion itself.¹ The diagnosis depends equally upon the presence of all three, and the absence of all others. If for instance any of the recti muscles are affected also, we are at once carried further back to the trunk of the third or sixth nerve; and if only one or two of the separate symptoms forming the triple group were present, the inference would be ready that the disease might be seated either in front of the ganglion or behind it in the trunk of the nerve involved. The ganglion itself is the only place where the two motor nerves (third and vaso-motor) are in contact, and where both may be affected by disease which does not involve any other parts.

¹ My only hesitation on this point is as to whether in some cases the same symptoms may be due to disease near the nucleus of the third nerve. It is not easy, however, in such cases to see why the vaso-motor should be affected. I have, however, seen the pupil motionless and accommodation lost in some cases in which the disease was believed to be in this position; always, however, there were other complications.

Clearly, then, it is easy to state beforehand what are the symptoms that ought to follow destructive disease of this ganglion, and to see that they could scarcely be caused by disease located anywhere else.

The second part of my task is to show that there are cases in which this precise group of symptoms is met with, and afterwards I shall have to make some suggestions as to the probable nature of the disease in question.

Of the three structures named it is probable that the circular or constrictor fibres of the iris are supplied solely by the third nerve, that its dilator or radiating fibres are innervated solely by the vaso-motor, and that the ciliary muscle although perhaps dependent in some degree upon both nerves for perfection of function, is chiefly supplied by the third. Any one of these muscular structures may be paralysed alone, and although combinations sometimes occur which it is difficult to interpret,¹ yet it is beyond doubt that the vaso-motor nerve may be wholly paralysed without cycloplegia, whilst the latter is a usual concomitant of dilated pupil due to paralysis of the third nerve.

The first case which drew my attention to this subject occurred in 1865, and was one of the most important that I have seen because the disease was slowly aggressive, and I was able to watch its several stages. The patient was a clerk (Mr. C—), æt. 34, when he first came under care. In the *left* eye the pupil was contracted owing to complete paralysis of the radiating fibres of the iris; he also had difficult accommodation with the same eye. He remained under my observation till 1868; at which date in the *left* eye iridoplegia was complete, and accommodation wholly lost,

¹ Although probably by far the greater part of the motor-nerve supply to the iris and ciliary muscle passes through the ganglion, it is not certain that these structures are wholly dependant on that body. Experiments in dogs make it seem probable that some vaso-motor twigs to the dilating fibres of the iris pass with the long ciliary, and never enter the ganglion. It may be so in man, and, within limits, the anatomical conditions may possibly vary in different individuals.

whilst in the *right* there was complete iridoplegia, but accommodation was still good. There was a remote history of mild syphilis, but although iodide of potassium was given no benefit followed, and the symptoms slowly advanced.

My next case exactly resembled the early stage of the one just recorded, but my notes do not extend over any long period, and I cannot therefore assert that the loss of accommodation ever advanced quite to completeness.¹ The patient, a gentleman, æt. 35 (Mr. E—), came under my observation in April, 1867. In his *left* eye the pupil was absolutely motionless and accommodation much weakened; with a + 15 lens he read No. 1 Jaeger easily. The other eye was perfect. He appeared to be in good health and denied all history of syphilis. He had suffered severely from sciatica. None of the recti muscles were defective. In a bright light the pupil of the paralysed eye was a little larger than that of the other, but in the shade this relation was reversed, the paralysed pupil remaining stationary while the other dilated.

CASE 3.—In April, 1869, a woman (Mrs. T—), æt. 44, who had been under my care for primary and secondary syphilis in 1865, but had in the interval been quite well, came to Moorfields on account of sudden failure of sight in both eyes. Her pupils were of moderate size, and so extremely sluggish that I was in doubt whether it could be said that they acted at all. She assured me that until three weeks before she could see to thread her needle perfectly, and that the loss of ability to do so had been quite sudden. With + 16 glasses she could just manage to read No. 1 Jaeger; her distant vision also was not quite perfect, in consequence perhaps of some small films which were present in the vitreous of each eye.

CASE 4.—*Complete symptoms of disease of lenticular ganglion in left, partial irido-plegia with weak accommodation*

¹ This case has been published in rather more detail in vol. vi of the 'Ophthalmic Hospital Reports,' p. 142.

in right.—Elizth. B—, an unmarried servant girl, who furnished the subject of this important case came under notice in June, 1873. In the *left* eye there was complete paralysis of the iris and ciliary muscle, whilst in the *right* the pupil was very sluggish, but the ciliary muscle was unimpaired. Not the slightest movement of the left iris could be provoked even by exposure to the strongest light. With a + 7 glass she read 1 Jaeger easily. It was probable from her statements that the symptoms had been slowly advancing for six months. She had suffered from much pain across the forehead. I did not ask the direct question as to syphilis, and I failed to detect anything about her in support of such a suspicion. Although in the *right* eye her power of accommodation seemed for the present nearly perfect, she said that the eye soon tired; and it was her impression that it was failing just as the other had done. This patient attended at Moorfields from June 19th to Oct. 2nd, and took throughout iodide of potassium in five grain doses. At the latter date she had so far improved as to be able with the *left* (paralysed) eye to read No. 4 Jaeger, although not easily. The pupil of this eye was still rather larger than that of the other (in a bright light). No further evidence had been obtained as to its possible cause.

CASE 5.—My fifth case is that of a gentleman, æt. 35 (Mr. T—), who discovered that his *right* eye was failing in 1861.

He consulted an eminent ophthalmic surgeon; his symptom being a certain inability to read whilst vision for distant objects remained good. This eye never regained its power of accommodation, and he was obliged ever afterwards to depend wholly upon the left for reading. Some time later the right eye began to diverge, and for troublesome diplopia he came under my care in April, 1869.

I found his *right* eye slightly divergent, its pupil absolutely motionless, and the function of accommodation lost. With a + 12 glass he read No. 1 Jaeger easily with the affected eye, and the sight of this eye for distant test-types was glass. It appeared that the superior,

inferior, and internal recti were all in a slight degree weakened. His other eye was not in any way affected, except that it presented a low degree of myopia. He denied any history of syphilis, and I could detect nothing suspicious. His face was covered with acne. He was subject to "rheumatic aching in the limbs." He had suffered from certain other nervous symptoms, thus, he was liable to a nervous cough which sometimes produced vomiting; he often had severe sick headaches; about a year ago he suffered an attack of most violent neuralgia in the face lasting a few days. These were the only items of evidence I could get in reference to his predispositions. It will be seen that the symptoms had lasted in the one eye for eight years, and that the other had not suffered in any degree. It appeared not improbable that disease beginning in the ganglion had travelled backwards to the trunk of the third nerve.

CASE 6.—Mr. H—, a veterinary surgeon, æt. 36, was sent to me on June 2nd, 1875, from a town in the midland counties. In him the condition was symmetrical, and in both eyes paralysis of the iris was complete and accommodation very feeble. He had had syphilis eight years before, and the details of his attack and treatment were supplied to me by the surgeon who attended him. He had no other symptoms of nerve disease excepting constipation, flatulence, and occasional stabbing pains in his limbs. I treated him for several months but without being able to prove any benefit. Sluggishness of pupils and occasional failure of sight had been noticed for a year before Mr. H— came under my care. When I first saw this patient he read 20 Snellen at twenty feet, but could only read large print without glasses. His optic discs were quite healthy, and his sight with low convex glasses was perfect for near objects. My notes do not state the precise degree of failure of accommodation, but at a subsequent visit he saw No. 5 Jaeger with one eye, and No. 8 with the other. His reason for applying to me was that he could not see to read or write efficiently. The cycloplegia was, however, neither complete nor constant;

thus, at one of his visits to me he managed with great effort to puzzle out a few words of No. 1 Jaeger. This was after some months' use of mercury and iodine, and after he had somewhat improved. His pupils remained throughout absolutely motionless; I tested them in sunlight and used a lens. The stabbing pains mentioned above had been severe enough to suggest the possible commencement of locomotor ataxy, but there were no symptoms conclusively indicative of that malady. He compared the pains to the pricks of a knife, and said that formerly they had occurred chiefly at night and would often keep him awake. His lower extremities were the parts usually affected. He got rid of these pains under iodide treatment, and is now, I believe, quite well.

Hitherto I have not been able to record in any single case that a cure has occurred. In the following instance, however, it seems highly probable that one of the three functions has been regained, since "paresis of accommodation" had been diagnosed four years before the patient came to me, and from this he had almost wholly recovered when I saw him, the iridoplegia, however, remaining complete.

CASE. 7. In which twelve years after syphilis the ciliary ganglion of one eye was affected, and recovery of the function of accommodation with permanent iridoplegia resulted.—This case is one of especial interest because I am able to feel certain as to the patient's antecedents, and also to state the result of treatment. The patient came under my care in May, 1871, and again on Oct. 8th, 1876.

Mr. S— was treated by Mr. H. J. Johnson, in 1860, for chancre followed by eruption, &c. He remained fairly well until 1870, when he was under Mr. Buxton Shillitoe's care for ulcers in the throat and on the tongue. At this time he also became deaf, and consulted Mr. Harvey. In May, 1871, he was brought to me by his medical attendant, Dr. Reilly, of Bow, on account of intractable syphilitic ulcers on the palate and tongue. I saw him only once, and he was subsequently under the care of different surgeons both in England and America for the same malady, always affecting

the same parts. In Oct., 1876, he returned to me having just come home from the Philadelphia Exhibition, and still suffering from sore tongue and lips. He now told me that his *right* eye had suffered about a year after his first visit to me, and that he had been under the care of one of my Moorfields colleagues on account of it. I found his *right* pupil of the same size as the other, but quite motionless whilst the left acted well. He could see almost perfectly in the distance with the *right*, but could not read so easily nor at so short a distance with it as with the other. His near point was eight inches with the *right*, and five inches with the *left*. He told me that when he applied to Mr. H— he could not see to read with the right eye at all though he could see with it in the distance, and that under treatment the sight of this eye had gradually improved. He considered that the eye was now quite well, and was not aware that the pupil was motionless. Mr. H—'s treatment had consisted in a six months' course of five-grain doses of iodide of potassium and the use of Calabar bean. I was shown Mr. H—'s hospital letter upon which "paresis of accommodation" was written as diagnosis; and on it was recorded that after the use of Calabar bean his near point with the *right* was eight inches, and that of the *left* five inches. It will be seen that we have no measure of the degree in which accommodation had failed, but Mr. H—'s expression in the note and the man's memory of his symptoms may be taken as evidence that the function was very considerably reduced.

Looking at the fact that the pupil is now motionless and neither contracted nor dilated, we may take it as highly probable that in the first instance all three functions were lost, and that the accommodation alone has been benefited by treatment. He has had no other symptoms of syphilis of the nervous system.

General summary.

Whatever may finally be thought as to the recognition of the precise part of the nervous system affected, it is apparent

that we have in the cases described in this paper illustrations of a very curious group of symptoms. The cases for the most part very closely resemble each other. In none of them was the patient very seriously ill, and in only two were there indications of disease of other parts of the nervous system. In none did the disease of the nervous system show any tendency to extend whilst the patients were under my observation, a fact, however, which may perhaps have been partly due to their having been all treated by specifics. Of the eight cases, in five both eyes were affected.¹ In three of the eight there was no history of syphilis (two men and one woman), but in none of these can the absence of history be held to prove absence of the reality. In two of these three only one eye suffered, the disease in one of them having been present eight years without showing any tendency to attack the other eye. All my patients were at an age when syphilitic affections of the nervous system are common; the oldest was forty-four, and the youngest twenty-seven when the disease began. In one case I had myself attended the patient for severe syphilis four years before the eye symptoms, and in four others there was the definite history that symptoms of constitutional syphilis had occurred a few years ago. I cannot but regard it as highly probable that in almost all cases the affection is due to syphilis.

We may note that it does not appear to occur amongst the later forms of tertiary disease, but rather during that period in which after disappearance of secondary symptoms there is usually a long period of latency.

One fact I must ask especial attention to; it is that the paralysis of the iris appears to precede the loss of accommodation, and also to resist treatment longer. In four cases it is expressly stated that the iridoplegia was first developed, and in two of them the patient regained to some extent the use of the ciliary muscle. In none was the failure of accommoda-

¹ As regards symmetrical occurrence the facts are very similar to what we find in the case of syphilitic choroiditis, in which frequently both eyes are affected, sometimes only one, and but rarely both in quite equal severity. The two conditions occur also at almost the same periods after the primary disease.

tion the first symptom, a fact the more important when we consider that it causes a condition which the patient is not likely to overlook, whereas a motionless pupil may easily escape attention. It is, I admit, difficult to explain, on the theory that the disease is in the ganglion, why the ciliary muscle and the sphincter of the pupil should suffer in different degrees.¹

It has been necessary in dealing with this somewhat intricate subject to use a number of terms some of which can scarcely be said to be in common employment. It may be convenient, even at the risk of some repetition of what I have just said, to define these and to add a few words about the conditions severally designated by each.

Paralytic mydriasis is the condition of dilatation of pupil which occurs when the circular fibres are paralysed and the dilators intact. It may result from disease in the short root of the ciliary ganglion, or in the trunk of the third nerve, or in the brain itself. When present alone it may be presumed to be due to disease in the short root. As an uncomplicated condition it is very rare.²

Paralytic myosis is the condition of contraction of the pupil which occurs when the radiating fibres are paralysed, the circular ones remaining intact. It results from disease of the vaso-motor root of the lenticular ganglion or from disorganisation of the trunk of the vaso-motor nerve in the neck, or from disease or injury to the brachio-cervical region of the spinal cord. It is seldom in such a case that the pupil is contracted in any marked degree, unless the circular fibres are thrown into spasm: a condition of simple inability to dilate but without any special contraction is the common result of paralysis of the vaso-motor. As a consequence of

¹ Is it not reasonable to suggest that the disease of ganglion acts at first by simply weakening the nerve currents which traverse it, and that a current which may be too weak to reach to the iris may still get as far as the ciliary muscle?

² When mydriasis occurs with cycloplegia, and without other complication, it may be conjectured that the third-nerve root of the ganglion is diseased. I have notes of several cases in which this diagnosis was given. I have never seen both eyes affected.

injuries to the cord or to the sympathetic trunk in the neck paralytic myosis is not uncommon, and is usually combined with narrowing of the palpebral fissure and retraction of the eyeball. When not so complicated it may probably be due to disease close behind the ganglion.

Irido-plegia is a term applicable to total paralysis of the iris, both of its circular and radiating fibres. It is usually an indication of disease in the lenticular ganglion, and is then commonly combined with cycloplegia. It may at times occur from disease in the centres, but when such is the case other combinations might be expected.

Cycloplegia denotes paralysis of the ciliary muscle, and is expressed by loss of power of accommodation. In young and middle-aged persons it is easily appreciated, the necessity of a strong convex glass for reading (+ 10 or + 12) being conclusive. Cycloplegia occurs often, without combination with other ocular paralysis, after diphtheria, but in all other conditions it is rare excepting with paralysis of the iris. It does not occur, or at any rate is not complete, when the vaso-motor trunk in the neck is paralysed. We may presume that the ciliary muscle is supplied almost solely by the third nerve, but perhaps partly by the vaso-motor.

Ophthalmoplegia interna is a term which may perhaps be conveniently used to indicate a state of paralysis of all muscular structures within the eyeball (both sets of fibres in the iris and the ciliary muscle), and excluding all the external or orbital muscles. It is the symptom, or combination of symptoms, with which the present paper is concerned, and when it is present without any addition I claim that in all probability it denotes disease located in the ciliary ganglion.

A few words, in conclusion, on the general bearings of the subject may perhaps be permitted. Pending the results of post-mortem dissection it must still remain doubtful whether the group of symptoms which I have described is due to disease in the peripheral ganglion or in some central nucleus the position of which is not yet known to anatomists. If we accept, as I think we may safely do for the present, the

hypothesis that the ganglion is its site, we have, then, tolerably conclusive evidence that it is possible for syphilis (and perhaps other causes of disease) to attack and disorganise the separate ganglia connected with the vaso-motor system. If the lenticular may be thus involved why not Meckel's or the otic, why not any one or any pair of the cervical or thoracic ganglia? Our task in the future must be to seek out and identify the groups of symptoms which diseases of each of the ganglia separately or of many together would produce, and it may easily be that here we shall find an explanation of some symptoms which have as yet been regarded as anomalous.

Another line of investigation in the future will be as to whether when disease has begun in a peripheral ganglion there is much tendency for the morbid process (probably a neuritis) to travel along the nerves and involve other trunks or even reach the central nuclei. In two of my cases something of this kind appeared to have happened, for the patients who began with ophthalmoplegia alone had subsequently paralysis of the third. In none others was there any tendency shown to invade other nerves, but as already stated it must be remembered that all the patients were treated with specifics. There is another group of cases which I hope to have the honour at an early opportunity of bringing under the notice of this Society in which all the external muscles of both eyeballs become either in succession or simultaneously partially paralysed, and for which I shall venture to propose the name *ophthalmoplegia externa*. This affection is usually due to syphilis, and in it probably the centres are affected. It curiously and unexpectedly happens that in it the *internal* muscular apparatus of the eye often escapes. In one case, however, which is included in the appended table, the internal paralysis in a complete form preceded that of the external muscles, but as a rule it is remarkable that the two affections, although due to the same cause, keep distinct.

A contracted and sluggish pupil is a well-known symptom of locomotor ataxy, and often an early one. It is probably caused by defect in the vaso-motor fibres, and is not usually

attended by failure of the ciliary muscles. In one or two of the cases which I have mentioned symptoms were present which suggested the possibility that ataxy was threatened, and in connection with this subject we must recollect that several French observers, M. Fournier most prominently, have adduced facts in favour of the belief that this malady is in many cases due to syphilis. My own experience quite supports M. Fournier's statements, but it is to be remembered that to both of us probably only special forms of ataxy have been presented. Whether or not ophthalmoplegia interna is often met with in association with ataxy must be left for further investigation, but the facts as at present known would not favour such a suspicion.

I must finally add a word of apology for the endeavour to introduce a new term into use. I am well aware of the inconveniences of a loaded nosology, but as I feel confident that the group of symptoms described is a definite and important one I feel justified in suggesting for it a distinctive name. Whether this course is desirable and whether the proposed name is a good one must be left to the decision of the future.

A tabular statement of the cases which have occurred in my experience is appended overleaf.

Table of cases of *ophthalmoplegia interna*.

No.	Name.	Age.	History as to syphilis.	One or both eyes.	Eye first affected.	Remarks.
1	Mr. C—,	34	Mild syphilis some years before	Began in one and afterwards affected both	Left	Iridoplegia preceded cycloplegia in each eye. (Notes extend over two years.)
2	Mr. E—,	35	No history of syphilis	One	"	Iridoplegia preceded cycloplegia. (Only a single observation.)
3	Mrs. T—,	44	Syphilis four years before	Both	Simultaneous	Iridoplegia and cycloplegia coincident.
4	Elizabeth B—, single	27	No history of syphilis	Left complete, right threatened	Left	Slight improvement under a course of iodide.
5	A gentleman	35	Ditto	One only	Right	The condition had persisted eight (? 18) years. Some weakness of third-nerve recti.
6	Mr. H—, Veterinary Surgeon	36	Had had syphilis eight years before	Both	Uncertain, perhaps simultaneous	Iridoplegia complete; cycloplegia partial and somewhat variable under treatment.
7	Mr. S—,	37	Had suffered severely from syphilis	One only	Right	Recovered from cycloplegia, the iridoplegia remaining.
8	A sailor	...	Syphilis mild—Both years before	Both	Simultaneous	With paralysis of all the eye-muscles. The lenticular paralysis had probably preceded that of the recti by several months. (I have not mentioned this case in the body of the paper.)

TREATMENT OF HÆMORRHAGE

FROM

PUNCTURED WOUNDS OF THE THROAT AND NECK,

ESPECIALLY CONSIDERED WITH REGARD TO LIGATURE
OF THE EXTERNAL CAROTID ARTERY.

BY

WILLIAM HARRISON CRIPPS, F.R.C.S.,
SURGEON TO THE GREAT NORTHERN HOSPITAL, ASSISTANT SURGEON TO
THE ROYAL FREE HOSPITAL.

(Received January 31st—Read April 23rd, 1878.)

THE violence of all bleedings in these localities, the suddenness of their occurrence, and the rapidity with which they prove fatal, surround this subject with grave interest. It is, indeed, in these cases of emergency that some of the most brilliant triumphs of surgery have been achieved. In ordinary incised wounds the treatment of hæmorrhage scarcely admits of discussion, ligature of the bleeding vessels *in situ* being the obvious treatment. It will be in the following circumstances that difficulties may arise.

1. Punctured wounds behind and below the angle of the jaw.
2. Punctured wounds through the mouth.
3. Hæmorrhage after removal of tonsils.

4. Hæmorrhage from cancer of the mouth and tongue.

5. Secondary hæmorrhage after wounds or surgical operations.

Fortunately in many of these cases the treatment is sufficiently clear. In some instances the wound may be enlarged with good prospect of securing the vessel. The careful and accurate adjustment of pressure may succeed. Cold and astringents have been usefully employed. In one or two cases the ingenuity of the surgeon has suggested a ready remedy, such, for instance, as in a case of secondary hæmorrhage after operating for cleft palate, in which Mr. Savory permanently arrested the bleeding by a wooden plug in the posterior palatine canal.

However successful these means have proved in many cases, there will remain a certain proportion in which their use has been employed in vain, or in which the very nature of the wound renders the application hopeless from the first.

The search for the wounded vessel is at times anatomically impossible, and even in accessible situations in secondary hæmorrhage it is a proceeding not unlikely to fail. In these cases surgeons have resorted to ligature of the main vessel in continuity, as the only practical means likely to prevent bleeding and prolong life.

The vessel almost universally selected is the common carotid, and the object of this paper is to investigate the facts bearing on this question.

In the appendix will be found a Table of fifty cases in which the common carotid has been tied for hæmorrhage, these cases being the result of a search through the English papers and reports, together with a few of the leading American journals. These cases have been in no way selected, and merely represent the total number found recorded. Many of these have been published in previous statistics, while some have not been previously tabulated. Statistics drawn from published records must always be received with caution, for successful cases probably find their way more readily into print than the failures. I think,

therefore, that the mortality in these should be taken as presenting a minimum rate. This suspicion is somewhat confirmed by finding that in hospital statistical reports in which all cases are recorded the death ratio is considerably higher. Out of the fifty cases collected twenty-eight died, or 56 per cent.

This mortality closely corresponds to that given in the tables of Pilz and Norris, and, I think, may be taken as fairly near the truth.

These tables show clearly that the operation of ligature of the common carotid artery must be classed amongst the most fatal in surgery.

The cause of this high rate of mortality will now be considered, and the following table shows the number of deaths arranged under five headings.

Causes of death in 28 cases.

Brain symptoms	.	.	.	8 cases.
Recurrent hæmorrhage from wound	.	.	.	8 „
Secondary hæmorrhage, seat of ligature	.	.	.	1 „
Dyspnœa	.	.	.	1 „
Exhaustion	.	.	.	5 „
Not stated	.	.	.	5 „
Total	.	.	.	28 „

Eight of these deaths, or 28 per cent., were directly consequent upon the operation and not attributable in any way to the accident for which the operation was performed.

Eight deaths took place in consequence of continued bleeding from the original wound after ligature of the common trunk, proving the futility of the operation; six cases remain, in which death probably took place from causes disconnected with ligature of the carotid artery.

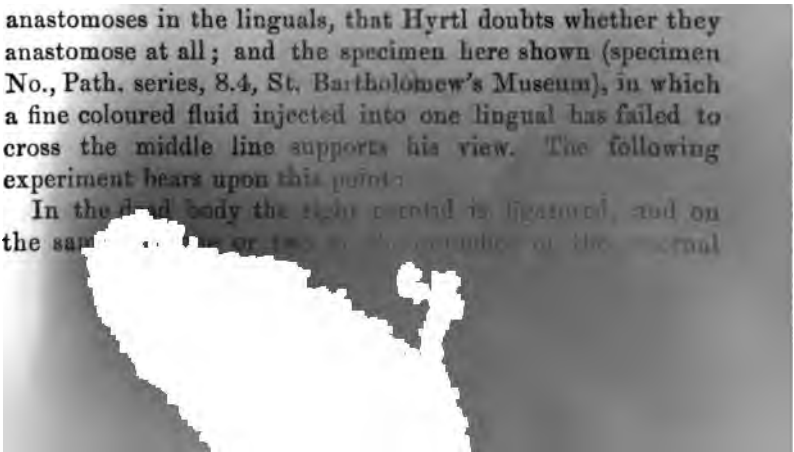
It is hardly within the province of this paper to consider in detail the cause of the brain symptoms, and for present purposes it is sufficient to state that interference with the
of the brain from obliteration of one of the internal

carotids is the direct cause of the phenomena. From Dr. James Russel's admirable papers in the 'Medical Times and Gazette,' 1874, it would appear that the brain symptoms are somewhat in proportion to the amount of blood lost previous to the operation, and that a brain already anæmic from general hæmorrhage is less able to support the sudden cutting off of the supply than one in a previously healthy condition. The second leading cause of mortality (recurrence of hæmorrhage from original wound) requires a more detailed consideration.

Assuming for reasons which will be subsequently explained, that the vessel originally wounded is the external carotid, or one of its branches, it becomes a matter of considerable importance to consider from which end of the wounded vessel the bleeding proceeds. Unfortunately in scarcely any of the cases collected has any note been made on this point. In two instances, however, it is stated that the blood came as a regurgitant stream from the proximal end. In the majority of instances, ligature of the common carotid temporarily arrested the bleeding; this could hardly have been the case had the blood come from the distal end. The following anatomical and experimental facts point to the same conclusion.

Blood obeying the same law as other fluids, will escape in the direction of least resistance. The large vessels at the base of the brain connecting the internal carotid both with its fellow of the opposite side and the vertebrals, would appear to offer less resistance to the blood current than is offered by the exceedingly fine anastomoses of the superficial vessels across the middle line. So fine are these anastomoses in the linguals, that Hyrtl doubts whether they anastomose at all; and the specimen here shown (specimen No., Path. series, 84, St. Bartholomew's Museum), in which a fine coloured fluid injected into one lingual has failed to cross the middle line supports his view. The following experiment bears upon this point:

In the dead body the right carotid is ligatured, and on the same side the carotid is divided, and the end of the external



carotid are divided; for example, the lingual and facial, at a distance of one inch from the main trunk. If water be now injected into the common carotid of the opposite side, it will be found that nearly all the water flows out through the proximal end of the divided vessel of the right side; that this arises principally through the anastomoses of the internal carotid as a regurgitant stream can be shown by placing a ligature upon the internal carotid of the right side, the effect of which is to cause the flow from the proximal end of the cut vessel almost entirely to cease.

The anastomoses of the superior thyroid appear to be freer than those of the other branches of the external carotid, and this is principally due to its free communications with deeper vessels of its own side. This vessel is probably an important channel of communication after ligature of the carotid. The following case mentioned by Guthrie bears upon this point:—"During an operation at Westminster Hospital, the external carotid was opened a little above its bifurcation. A ligature was placed upon the common carotid; the bleeding was not in the least arrested. A ligature was then placed upon the external carotid above the wound; it still continued to pour out blood. A third ligature was placed upon the internal carotid without success. The external carotid was then ligatured just below the wound, but beyond the superior thyroid, when the bleeding ceased.

Notwithstanding the fact that in the majority of instances the bleeding takes place through the proximal end, it seems probable that in some cases it may be the distal end of the wounded vessel that furnishes the blood. Except upon this hypothesis it is difficult to explain the phenomenon that has been especially mentioned in three cases (Nos. 4, 41, 50), namely, that when the ligature on the common carotid was tightened, an increased gush of blood immediately took place from the wound.

Something might be ascertained in the way of diagnosis by observing the effect of pressure upon the carotids of either side.

large proportion of the deaths are

directly due to brain symptoms caused by ligature of the common carotid, and seeing how often the operation has proved futile in preventing death by bleeding from the original wound, it would appear that in the face of these facts most cogent reasons would be required to justify its continuance. This justification could only be found in the firm belief that surgery offered no other resource to avert an inevitable death. Such is not the case, for in ligature of the external carotid will be found an effectual alternative to the operation upon the common trunk.

The objections raised to this operation are as follows :

1st. The fear of secondary hæmorrhage from the seat of ligature due to close proximity of its larger branches.

2ndly. The futility of the operation should the wounded vessel be the internal carotid.

3rdly. That it is less easy to ligature than the primitive carotid.

The fear of secondary hæmorrhage must be a theory entirely grounded upon the anatomical arrangement of the carotid and its branches, and disappears altogether before the test of actual experience.

M. Guyon, in 'Mémoires de la Société de Chirurgie,' vol. vi, mentions twenty-seven cases in which the external carotid had been ligatured.¹ Some of these occurred in wounds, others either in continuity before surgical operation, or on accidental section during their performance. To this number I can add three cases. In only a single instance did secondary hæmorrhage occur. Thirty cases are doubtless too small a number upon which to venture any accurate estimate as to the frequency of this accident. But that it only occurred once in thirty times is evidence that the external carotid may be reckoned as amongst the least liable of all the large vessels to bleeding at the seat of ligature.

To the second objection the answer lies in an appeal to the fact of how rare is a wound of the internal carotid compared

¹ Amongst these are included cases by Maisonneuve and Chassaignac, fuller details of which will be found in the 'Bulletin de la Société de Chirurgie,' Tom. I.

with that of the external or its branches. In eighteen cases in which the bleeding vessel was identified, the following were the vessels wounded :

Common carotid at point of bifurcation .	2
External carotid .	2
Lingual .	1
Facial .	1
Tonsillar branch .	1
Branch in parotid gland .	1
Ascending pharyngeal .	1
Internal maxillary .	2
Inferior dental .	1
Middle meningeal .	1
Vertebral .	1
Internal carotid .	2
External also wounded .	1
Close to bifurcation .	1
Total .	18

The internal carotid was found only to have been wounded twice alone, and once in conjunction with the external. In many cases of wounds and operations about the tonsils the internal carotid was supposed to have suffered, but in the majority of instances the vessel had proved to be a branch of the external carotid. It cannot be denied, however, that wounds of the internal carotid have occurred, and that in these cases a ligature on the external carotid would be useless. In the event of the mistake occurring it is not beyond remedy, for a ligature might still be placed upon the internal at the bifurcation of the common carotid. On the other hand no remedy can be found for a patient dying with hemiplegia caused by obstructing the internal carotid on account of a wound of the external.

Taking this into consideration, and bearing in mind the relative frequency of either mistake, I do not think it can be urged as a serious objection to ligature of the external carotid.

The last objection, that the vessel is more difficult to find, requires no comment.

In favour of the operation the following reasons may be powerfully urged :

1stly. That circulation through the brain is not the least interfered with. Consequently, one large element of danger is avoided.

2ndly. That the danger of recurrent hæmorrhage from the wound must be diminished in proportion to the number of cases in which it occurs from the proximal end, as a regurgitant stream through the internal carotid or through the superior thyroid.

3rdly. That the incision made over the external carotid will also expose the bifurcation and internal carotid, and may thus lead to a direct exposure of the wounded vessel. (See Cases Nos. 4, 7, 18).

It now remains to be considered upon what portion of the external carotid the ligature can be best applied. From a considerable number of measurements I find that, in about half, the superior thyroid arises almost immediately after the bifurcation of the primitive trunk, and very rarely at a greater distance than half an inch. In most bodies will be found an interval from half an inch to three quarters of an inch between the origin of the superior thyroid and lingual, and a point situated midway between these vessels offers the best site for ligature. If during the operation the lingual appears low down in the wound, probably the safest course would be to place a ligature around it.

In conclusion I would state, that this paper is not meant to advocate ligature of the external carotid artery as an ordinary operation for hæmorrhage, but as a substitute for that of the common carotid, and as a procedure only to be had recourse to in cases of emergency, after all simpler means have been tried in vain.

Table of fifty cases in which the common carotid was tied for a wound in the throat or neck.

Reference.	Operator.	Cause of operation.	Cause of death.	Result.	Vessel wounded.
Lancet, 1850, vol. ii, p. 110	Luke	Cut throat	Bleeding recurred many times; death 30 days after last bleeding; became delirious and died Death 3rd day	D.	External carotid.
Lancet, 1854, vol. i, p. 664 Lancet, 1864, vol. i, p. 90	Critchett Semmes	Hæmorrhage from facial artery from sloughing Gunshot wound	Left carotid tied for secondary hæmorrhage; bleeding recurred six days later. The right carotid was then tied. Patient died 30 hours later	D.	—
Lancet, 1850, vol. ii, p. 143	Guthrie	Wound just at point of bifurcation; secondary hæmorrhage	Ligature failed to arrest the bleeding, but was stopped by one on external carotid beyond the wound; died next day	D.	At point of bifurcation.
Lancet, 1864, vol. i, p. 659	Partridge	Penetrating wound behind angle of jaw; secondary hæmorrhage on sixth day; carotid tied	Frequent bleedings from original wound	D.	Large branch in parotid gland, the external carotid being untouched.
Lancet, 1864, vol. ii, p. 523 Lancet, 1846, vol. i, p. 134	H. Lee Vincent	Hæmorrhage from sloughing of cervical gland Punctured wound of fauces	Death in 16 hours	D.	—
Lancet, 1846, vol. i, p. 135	Narrated by Arnott	Punctured wound through the mouth; secondary hæmorrhage	Death 9 days later, with recurrence of hæmorrhage Hæmorrhage recurred, and the patient died, but not as an immediate consequence of bleeding	D.	Vessel wounded just at bifurcation of common carotid.
				D.	Internal carotid <i>supposed</i> to have been wounded.

No.	Reference.	Operator.	Cause of operation.	Cause of death.	Result.	Vessel wounded.
9	Med. Times & Gaz., 1856, vol. i, p. 209	Hutchinson	Bleeding from cancerous ulceration	Brain symptoms; died on 7th day	D.	—
10	Med. Times & Gaz., 1858, vol. i, p. 89	Cooté	Bleeding from malignant tumour of antrum	Paralysis; died in 36 hours	D.	—
11	Med. Times & Gaz., 1864, vol. ii, p. 541	Poland and Beeby	Bleeding from cancerous mass	Paralysis on left side; died in 3 days	D.	—
12	Med. Times & Gaz., 1859, vol. ii, p. 429	Stanley	Secondary hæmorrhage; penetrating wound of fauces	Died; hæmiplegia 2 months later	D.	Internal carotid was not wounded; it was supposed to be the tonsillar branch.
13	Med. Times & Gaz., 1873, vol. i, p. 113	Wagstaffe	Secondary hæmorrhage after removal of superior maxilla	Gradually died from exhaustion on 4th day	D.	—
14	Med.-Chir. Trans., vol. viii, p. 224	Brodie	Hæmorrhage after extraction of tooth	Hæmorrhage recurred; hæmorrhagic diathesis	D.	Inferior dental.
15	Med.-Chir. Rev., 1827, p. 165	Travers	Wound behind angle of jaw	Bleeding frequently recurred on the last occasion to the extent of 2 lbs., when he died	D.	Wound just at commencement of facial; all the lower part of common carotid plugged, upper open.
16	Amer. Journ., 1846, p. 478	Sédillot	—	Complete hæmiplegia	D.	—
17	Amer. Journ., 1864, July, p. 27	Keen	Secondary hæmorrhage from wound	Recurred several times after ligature; a month later total paralysis of right side	D.	Internal maxillary wounded.
18	Amer. Journ., 1848, Jan., p. 866	Fraser	Punctured wound, angle of jaw	Died from extreme exhaustion	D.	Internal carotid wounded $\frac{1}{4}$ an inch from its origin.
19	Brit. Med. Journ., 1862, vol. ii, p. 489	—	Secondary hæmorrhage from injury to internal maxillary	Secondary hæmorrhage from seat of ligature	D.	Internal maxillary.
20	Brit. Med. Journ., 1863, vol. i, p. 399	Prichard	Punctured wound of neck	Hæmorrhage recurred	D.	Vertebral.
21	St. Barth. Hosp. Rep., 1876, p. 163	Baker	Secondary hæmorrhage after punctured wound of fauces	Died a few hours after operation	D.	Ascending pharyngeal.

		Savory	Hæmorrhage from a cyst in neck that had been previously opened Punctured wound Cut throat	Never recovered sensibility after operation	D.	External carotid.
22	St. Barth. Hosp., 1871					
23	St. Barth. Hosp.	Holden		Not stated	D.	—
24	Dublin Hosp. Rep., vol. iii, p. 336	Cussack		Bleeding continued with undiminished violence, and in a few minutes death took place	D.	—
25	Abernethy's Surg. Oper., vol. ii, p. 115	Abernethy	Wound of neck	Brain symptoms	D.	Internal carotid and branches of external wounded.
26	Edin. Med. Journ., vol. lxii, p. 126	Duncan	Hæmorrhage from fauces	Sudden dyspnoea	D.	—
27	Birmingham, Times, March 27th, 1875	—	Secondary hæmorrhage, punctured wound angle of jaw	Died in a comatose condi- tion a few hours after operation	D.	Middle meningeal.
28	Report of Army in Crimea, vol. ii, p. 308	—	Primary hæmorrhage	Died 4 hours after operation	D.	External carotid wounded.
29	Lancet, 1835, vol. ii, p. 437	Ellis	Secondary hæmorrhage; punctured wound angle of jaw	—	R.	Remarks. Doing well after 3 weeks.
30	Lancet, 1847, vol. i, p. 153	Le Gros Clark	Punctured wound	—	R.	—
31	Lancet, 1849, vol. i, p. 556	Eves	Wound of throat	—	R.	—
32	Lancet, 1850, vol. ii, p. 118	Johnson	Punctured wound of fauces	—	R.	—
33	Lancet, June 22nd, 1872	Pearse	Sudden hæmorrhage to the amount of some pints from sloughing of fauces	—	R.	Doing well 2 months later.
34	Lancet, 1853, vol. ii, p. 225	Thomas	Wound of temple	—	R.	—

No.	Reference.	Operator.	Cause of operation.	Cause of death.	Result.	Remarks.
35	Lancet, 1870, June 11th	W. Baker	Violent secondary hæmorrhage after removal of tumour situated over the external carotid	—	R.	—
36	Lancet, 1859, vol. i, p. 559	Ure	Punctured wound of fauces, some pints of blood lost	—	R.	Two or three slight recurrences of bleeding; was doing well 9 days after operation.
37	Lancet, 1850	Twitchell	Gunshot wound	—	R.	External and internal; the ligature did not arrest the hæmorrhage, which had to be controlled by pressure.
38	Lancet, 1850, p. 111	—	Punctured wound angle of jaw	—	R.	Lingual and thyroid supposed to be wounded
39	Med. Times & Gaz., 1858, vol. ii, p. 217	Field	Secondary hæmorrhage after removal of superior maxillary	—	R.	Doing well 6 weeks later
40	Med. Times & Gaz., 1859, vol. ii, p. 343	Chaussagnac	Aneurism at the back of throat punctured in mistake for an abscess	—	R.	—
41	Med. Times & Gaz., 1860, p. 190	Le Gros Clark	Wound of fauces	—	R?	Immediately after ligature was tied a gush of blood took place from the original wound; this ceased with pressure; doing well 1 month after operation
42	Med. Times & Gaz., vol. i, p. 90	Dewar	Bleeding from pulsating tumour of fauces	—	R.	—
43	Med. Times & Gaz., 1855, vol. i, p. 32	Winchester Hospital	Hæmorrhage from temporal artery	—	R.	—
44	Med. Times & Gaz., 1865, p. 385	At Oxford	Hæmorrhage from mouth	—	R.	—
45	Med.-Chir. Trans., vol. vii, p. 107	Collier	Punctured wound of mouth	—	R.	—

46	Med.-Chir. Trans., vol. xxii, p. 135	Scott	Hæmorrhage from nose; no wound	—	R.	—
47	Med.-Chir. Rev., 1827	Fleming	Wound	—	R.	—
48	Amer. Journ., 1864, April, p. 334	McCullough	Secondary hæmorrhage	—	R.	—
49	Amer. Journ., 1861, April, p. 601	Curtis	Secondary hæmorrhage; gunshot wound of mouth	—	R.	—
50	Dublin Hosp. Rep., vol. i, p. 301	Browne	Punctured wound below angle of jaw; secondary hæmorrhage	—	R.	On first tightening the ligature hæmorrhage re- curred as violently as ever; stopped by pressure. Had one or two attacks of convulsions, but re- covered.
51	Amer. Med. Times, 1861, April 20th	J. C. Hutchinson	Secondary hæmorrhage; wound of internal maxil- lary	—	R.	



A CONTRIBUTION
TO THE
PATHOLOGY OF HÆMOPHILIA.

BY
PERCY KIDD, B.A. OXON.
DEMONSTRATOR OF PRACTICAL PHYSIOLOGY AT ST. BARTHOLOMEW'S
HOSPITAL.

(COMMUNICATED BY DR. SAMUEL GEE.)

(Received January 24th—Read May 14th, 1878.)

THE comparative rarity of cases of hæmophilia and the great interest attaching to this disease, are the grounds on which I venture to bring before the Society the following short paper. Although a certain number of "post-mortem" examinations have been made on cases of hæmophilia, very few have been supplemented by a microscopical examination, and in these I believe the results have been negative. The conclusions which I wish to put before the Society concerning the pathology of this disease are unfortunately drawn from a microscopical examination of one case only.

Before proceeding to describe the pathological changes that have occurred, I will first give a brief account of the history of the case :—

William D—, æt. 6 years, was admitted into the Hospital for Sick Children, London, on August 31st, 1877, under the

care of Dr. Gee (to whom I am indebted for permission to publish the case).

His state on admission is described thus :—A very anæmic, pale child, but not thin. Complexion distinctly greenish mucous membranes white. Is now bleeding from the gums. The blood comes partly in clots, partly in a liquid state, and is very watery. No obvious lesion to account for the bleeding. Spleen not enlarged. Glands not enlarged, with exception of one in right groin, which is slightly increased in size. No purpura. Urine acid, no albumin or blood in it. Heart's action very rapid. An ill defined systolic apex murmur.

Previous history.—Has had no specific fever. Was a stout child with a good colour till two years old. When two years old he struck his head against a wall ; the skin was not broken, but the bruised place swelled to the size of an egg, and became purple. At the same time he had considerable epistaxis, which was difficult to check. Had to be taken to the hospital. Since then has bled from nose and mouth about every two months. Bleeds greatly if he cuts himself. The worst bleeding was from a cut ten days ago. Though this was little more than a scratch he bled for a week. No blood in his urine at any time. Motions very black the last week, as is often the case when he cuts himself. Appetite poor usually. Not often sick. Yesterday he vomited a little clotted blood ; this often happens when his mouth bleeds. Never had a cough or hæmoptysis. Has been bleeding from the mouth the last ten days. The youngest child of the family, aged eight months, has been ill for a fortnight with diarrhœa, but not feverish. Yesterday bled from the bowels about $1\frac{1}{2}$ oz. Is not a pale child, and is considered healthy. It was subsequently ascertained that this child also died of hæmorrhage a few days later. After his admission the child was very drowsy and depressed, and became blanched to the last degree. The pulse was very feeble. The gums continued to bleed.

On September 3rd the child was ordered some turpentine every four hours, but died on September 4th. The

last thirteen hours before death the hæmorrhage ceased, but the child sank gradually. There was no diminution in the amount of urine, and the urine contained no blood. The blood from the gums was examined, and showed a large excess of colourless corpuscles, viz. 1 to 3 red ones.

Examination eleven hours after death.—Rigor mortis very strong. Weight $28\frac{1}{2}$ lbs. Body contained a considerable amount of subcutaneous fat. Muscles pale. Lungs remarkably pale and bloodless, even to their bases. No adhesions. No subpleural hæmorrhages. A good deal of œdema of lungs. Some clear fluid in pleural cavities. Right lung weighed $6\frac{1}{2}$ oz.; left lung $4\frac{3}{4}$ oz. Pericardium natural.

Heart.—Left ventricle decidedly fatty for almost half the thickness of its wall, the inner half of muscoli papillares showing very decided yellow lining and spotting. Valves natural. Very pale clot interlaced in columns of right ventricle and auricle. Blood remaining looks almost like water.

Aorta remarkably thin. Iliac veins and arteries also thin. Lining membrane natural. Liver $20\frac{1}{2}$ oz. Spleen $1\frac{3}{4}$ oz. Kidneys $3\frac{3}{4}$ oz., all pale and bloodless, but not fatty, and showing no change. Right knee-joint examined and found natural. Mesenteric glands a little enlarged. Bronchial glands not enlarged. Spleen pale and flabby.

The parts examined microscopically were the aorta and vena cava superior, and a piece of the mucous membrane of the mouth close to the entrance of the parotid duct where the fatal bleeding took place.

The tissues were hardened in bichromate of ammonia 2%, and sections were stained with hæmatoxylin and picrocarmine, and mounted in Canada balsam. Fortunately the parts were obtained in a relatively fresh condition, and were remarkably well preserved, so that "post-mortem" changes were as far as possible avoided.

I will first describe the condition of the mucous membrane of the mouth, and then the changes in the large blood-vessels:

Mucous membrane of the mouth.—For purposes of com-

parison I have given a drawing of the laminated epithelium which covers the mucous membrane of the mouth normally (see fig. 1, Plate XIV). This section was taken from a part of the mucous membrane where the surface epithelium was unaffected. The epithelium is seen to be divided into—
1. A superficial part, where the outlines of the cells cannot be made out, and the nuclei are indistinct. 2. A deeper part, where the cell borders and nuclei are quite sharply defined. Into this part the papillæ of the submucous tissue are seen to project. The condition of this epithelium is quite sufficient to show that the tissue has been well preserved, and has undergone no appreciable "post-mortem" change. The surface epithelium generally in nearly all the portion of mucous membrane examined was in a similar healthy state. But over a small irregular area, whose greatest diameter was rather less than $\frac{1}{8}$ th inch, the epithelium was profoundly altered.

In fig. 2 this condition of the epithelium is represented. The epithelium is seen to be reduced somewhat in thickness, and has an indistinct, dirty, and if we may use the term, "smudgy" appearance. In only a few places can any nuclei be distinguished, and throughout there is hardly a trace of the outlines of the cells. This degenerate epithelium resembles somewhat the superficial layers of the normal epithelium, but differs from it in possessing no nuclei, or at least very few, and in its more indistinct, glassy appearance. This is well seen by comparing fig. 2 with fig. 1, Plate XIV. The deeper parts of the epithelium were much more altered than the superficial layers. In both these positions, but especially near the surface, the epithelium is traversed here and there by irregular horizontal fissures, as if it had become separated into layers. This indistinct epithelial tissue was unaffected by staining reagents. The papillæ were either altogether absent, or, if present, were very much atrophied. Some of these atrophic papillæ were very short, others nearly the normal height, but in each case it was rare to find in them any trace of capillaries.

It is evident that in this part the surface epithelium has

undergone a necrotic change or degeneration, the individual cells becoming apparently fused together into an indistinct-looking, glassy mass. On either side of this necrotic area which was never longer than $\frac{1}{8}$ th inch, the degenerate material passed gradually into normal epithelial cells. In fact, in every section where this necrotic patch was visible, it happened that it was situated at the centre of the section, and at either end of the section it was always possible to find perfectly normal epithelium with distinct nuclei. The submucous tissue beneath this degenerate epithelium was very poorly supplied with blood-vessels, and the few vessels that it contained showed the same changes as those of the rest of the submucous tissue, which I will now describe.

The submucous tissue itself presented no change, but its arteries, capillaries, and veins, all showed considerable alterations. In each case the endothelial cells lining the vessels had undergone an extensive proliferation, some vessels showing this change to a more marked extent than others. The vessels most affected in this respect were the small veins. In some of these the endothelial proliferation was so great as to block up the vessel more or less. The vessels of the striped muscles lying beneath the submucous tissue were similarly affected, so that one description will suffice for the intermuscular vessels and those of the submucous tissue.

A good example of this condition of the endothelium is seen in fig. 4, which shows a longitudinal section of a small intermuscular vein containing an immense number of endothelial nuclei. The capillaries showed a similar proliferative change, whether situated in the submucous tissue or in the muscles (see figs. 3 and 4). In the papillæ bordering on the necrotic area, the capillaries were slightly different in appearance. Here the endothelial nuclei were jagged and irregular, but had also evidently undergone proliferation.

This would seem to suggest that the atrophic condition of the papillæ, as shown by the degenerate appearance of the endothelial nuclei of these capillaries, as well as by the diminished size of the papillæ themselves, was a later change

than, or possibly the result of, the alterations in their vessels. For it could hardly be supposed that proliferation would occur in the capillaries of papillæ already more or less atrophic.

In fig. 3 *c, p*, Pl. XIV, there is a drawing of such a capillary.

In fig. 5 *a, a'*, are two small arteries from the submucous tissue similarly affected, though here the proliferation is not so luxuriant as in the small veins. This condition of the arteries was seen in most cases. But in certain number of arteries a further pathological condition was observed, which is represented in figs. 5 *b, b'*. In these cases, in addition to the above changes, it is seen that the muscular coat, has a peculiar appearance. Instead of a regular circular coat of muscular fibres, these arteries have thick walls, in which there are hardly any distinct muscular elements.

On comparing these arteries with the other two in fig. 5 *a, a'*, which are fairly healthy as regards their muscular coat, it is evident that the normal spindle-shaped muscle nuclei, visible in the one case, are replaced here by larger and more irregularly disposed nuclei, many of which are constricted and lobed as if they were undergoing division. These large nuclei must be regarded as muscle nuclei, some of which are proliferating.

The coats of these arteries are mainly composed of an indistinct, slightly opaque tissue, without any definite structure, apparently resulting from a hydropic degeneration of the muscle fibres. In most arteries showing this change all distinction between muscular coat and "adventitia" is lost; but in some, the boundaries of the two coats are faintly visible. The calibre of all these arteries is greatly diminished, owing to the swollen state of their muscular coat. Examples of this form of arterial degeneration were found in the muscles, and in the submucous tissue, sometimes close under the epithelium. A careful examination of the vessels of the submucous tissue hardly ever failed to discover nuclei that were deeply constricted or lobed, and seemed to be in the very act of division.

The condition of the aorta and vena cava can now be dismissed very shortly. The walls of these large vessels themselves were quite normal, their lining endothelium being unaffected. But in their vasa vasorum, small arteries, veins and capillaries, endothelial proliferation was well-marked.

In fig. 6, Ao, are small vessels from the adventitia of the aorta, and those marked V. C. are vessels from the coats of the vena cava. With the exception that the small arteries do not show any hydropic change, these small vessels differ in no respect from the others already described. I have not been able to discover any further changes in the parts which I have examined. It may seem rash to speak at all positively from an examination of one case; but nevertheless, it does seem clear that we have to do here with a general affection of the small vessels, particularly of the smaller veins. A similar condition has been observed in scarlatina by Dr. Klein in the small vessels of certain organs, but, with this exception, I am not aware that such a general lesion of the vessels has been described in any disease. In estimating the importance of this proliferative change, the age of the patient should no doubt be taken into account.

But though we should expect to find evidence of a more or less active growth in the tissues of a child six years old, we cannot account for such an exaggerated activity of the vascular endothelium by a reference to normal processes. The importance of such a change in the vascular wall in the production of hæmorrhage is evident, and need not be further dwelt upon. Although I had the opportunity of examining the above parts only, I think it may be assumed that the vascular affection is a general one. For the coexistence of a similar condition of the small vessels in parts so far removed from each other in every way as the mucous membrane of the mouth and the large vessels of the chest, seems to justify such a conclusion.

Of the relation of the endothelial proliferation to the hydropic change in the comparatively small number of arteries so affected, I am not in a position to speak with any

But it would seem rational to suppose that the

endothelial change was the primary one from the condition of the other vessels.

The increase of the colourless corpuscles and the watery state of the blood show that the blood itself was affected, though these conditions may have been the direct result of the bleeding. But these considerations do not enable us to determine whether the blood itself or the vessels are primarily at fault. It is difficult to speak with any confidence with regard to the peculiar affection of the epithelium in the small necrotic patch shown in fig. 2. If the change in the vessel be considered to be the primary one, it is quite possible that any slight injury, such as might happen during mastication, would be sufficient to determine a destructive change in the epithelium of a part whose vessels were already diseased. Or it may be, that the affection of the epithelium was the direct result of the disease of the vessels which was most advanced in this particular spot. It is quite certain that the submucous tissue here was remarkably devoid of vessels, the few that it did contain were all affected with the proliferative change, and were more or less compressed.

I have dwelt upon this condition of the epithelium, because it seems possible that this necrotic patch may have been the point at which the blood escaped. The existence of gaps or fissures in this degenerate epithelium suggests the possibility that these were caused by the blood forcing its way out towards the surface through this tissue, and so causing these appearances. One would expect to find some blood-corpuscles arrested in the epithelium, but in no case did I find any blood-corpuscles in the epithelium. It should be remembered, however, that there was no bleeding during the last thirteen hours of life.

I have not attempted to review the various theories as to the pathology of this disease, as a full account is given of these in Dr. Legg's 'Treatise on Hæmophilia.'

My object has been to lay before the Society shortly and without reference to any theory, the changes that I have observed in the tissues in this disease.

DESCRIPTION OF PLATE XIV.

Pathology of Hæmophilia. (Percy Kidd, B.A.)

FIG. 1. Normal laminated epithelium of mouth. *a.* Superficial part. *b.* Deeper portion containing papillæ (*p*). Hartnack, 3·4 = × 90.)

FIG. 2. Necrotic epithelium. No distinct separation into superficial and deep layers as in Fig 1. *p.* Atrophic papillæ. *f.* Fissures in epithelium. (Hartnack, 3·4 = × 90.)

FIG. 3. Small vessels from submucous tissue. *v.* Small veins. *c.* Capillaries. *c.p.* A capillary from one of the papillæ bordering on the necrotic area, showing angular, jagged, endothelial nuclei. (Hartnack, 3·7 = × 300.)

FIG. 4. *v.* Small intermuscular vein, showing great endothelial proliferation. *m.* Striped muscle cut transversely. *c.* Intermuscular capillaries, also showing an increased number of endothelial nuclei. (Hartnack, 3·7 = × 300.)

FIG. 5. Small arteries from submucous tissue. *aa'.* Showing merely proliferative change in their endothelium. *bb'.* In addition to this change, showing a hydropic degeneration of their muscular coat and a proliferation of their muscle nuclei. The division of muscle nuclei is particularly well seen in *b*, which is a transverse section. (Hartnack, 3·7 = × 300.)

FIG. 6. Small vessels from the coats of the aorta and vena cava. *4a.* Small vein and capillary from aorta. *v.c.* Small artery, vein and capillary from the vena cava. (Hartnack, 3·7 = × 300.)

N.B.—The appearance of the original specimens and drawings has been somewhat misrepresented in all the lithographed figures. In these plates the outer coats of the vessels and the surface epithelium are distinctly striated, whereas scarcely a trace of striation is visible in these structures in the originals.

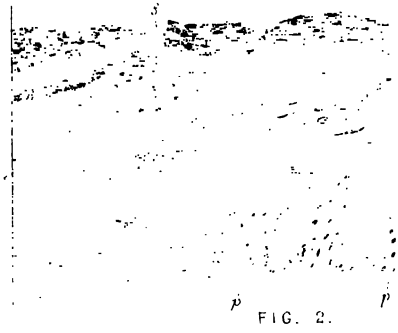
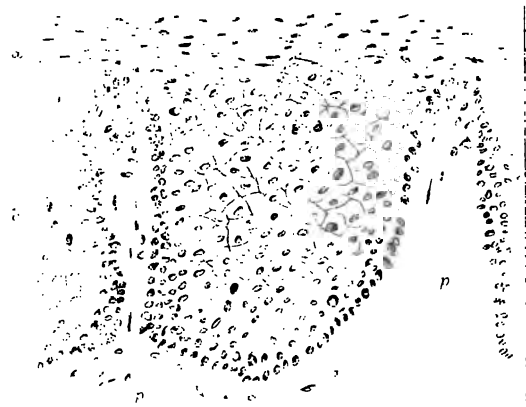


FIG. 2.

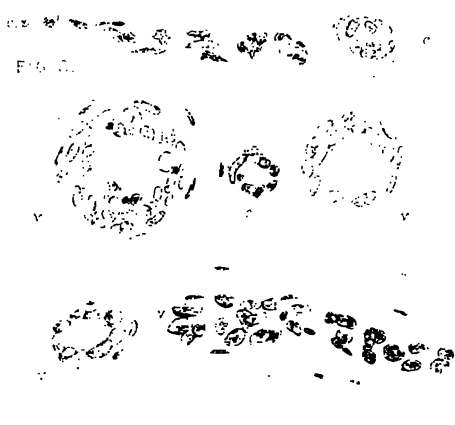


FIG. 3.

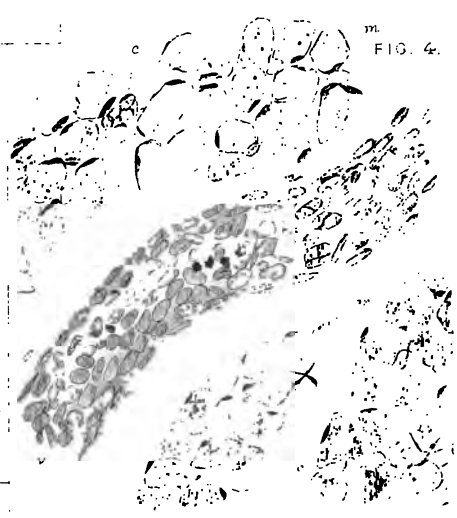


FIG. 4.

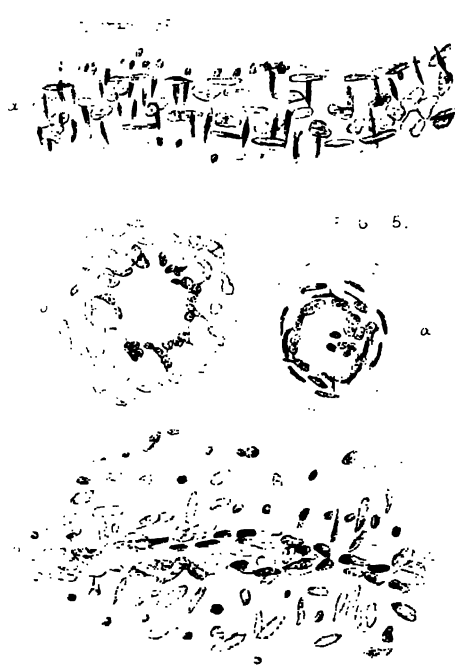


FIG. 5.



FIG. 6.



ON
THE PATHOLOGICAL TRACES
OF
PULMONARY HÆMORRHAGE.

BY
REGINALD E. THOMPSON, M.D., F.R.C.P.,
SENIOR ASSISTANT PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION,
BROMPTON; AND CURATOR OF THE PATHOLOGICAL MUSEUM.

(Received February 12th—Read May 14th, 1878.)

THE relation of pulmonary hæmorrhage to pulmonary phthisis is a subject which has always, even from the time of Hippocrates, attracted the attention of physicians, and the possibility of a phthisis *ab hæmoptoe*, firmly held in old times, concisely and clearly urged by Morton and Hoffman, denied by Laennec, has again in modern days received fresh confirmation in the well-known opinions put forward by Niemeyer in his writings on pulmonary phthisis.

On searching the evidence brought forward to sustain or controvert this theory, it is a little surprising to find a remarkable paucity of pathological proof of the effects of hæmoptysis, notwithstanding the great frequency of such an occurrence in cases of phthisis; much reliance appears rather to have been put on the clinical histories and symptoms of cases.

Valuable, however, as such a method of investigating the

question may be taken to be, I am inclined to believe, after considerable experience in the clinical aspect of the matter, that it is from the pathological point of view that the solution of a most difficult and intricate problem must be hoped for; and it is with the object of supplementing the pathological evidence which bears upon the subject of pulmonary hæmorrhage, that the following results, obtained from the examination of many cases in which hæmoptysis was known to have occurred, and in some of which the hæmorrhage was the immediate cause of death, are put forward as some contributions to the elucidation of a question which is as interesting as it is complex.

The opportunities afforded in the pathological department of the Hospital for Consumption, at Brompton, are considerable; and I must here express my thanks to my colleagues, Dr. Pollock, and Dr. Douglas Powell, who have with most courteous readiness placed many cases under their care at my disposal.

The details of a few of the cases which have furnished material for the conclusions which are here given are supplied in the form of an appendix, as it seemed advisable not to obstruct the general results obtained from their consideration.

The first question that requires an answer is this:—What traces are most frequently found in the lungs after death which indicate a previous hæmorrhage? There are certain peculiar pathological conditions which are often to be found in cases in which there has been a history of hæmoptysis, and they occur in no other instances. These consist of well-defined circumscribed nodules of an oval or round form, in colour varying, according to their age, from blood-red to yellowish-red and white, in size varying from $\frac{1}{8}$ of an inch to an inch, but generally assuming the size of a threepenny piece, say half an inch in diameter, marked in their centre with the openings of two or more bronchioles, which are usually surrounded or spotted with pigment; they have a slightly granular surface when red, but when white are firm, tough, unyielding, and hard (Plate XV, fig. 2 and 3). These have

usually been held to be simply nodules of caseous pneumonia, and are recognised as such in the cases recorded by Niemeyer, Bäumlér, Weber, and Powell. If I do not misunderstand the opinions of these observers, they consider these nodules to be the caseous results of a pneumonia, due to the irritation of blood remaining behind in the alveoli acting as a foreign body.

The opinion of Hoffman (1740), quoted by Niemeyer in his lectures on phthisis (pp. 30), that blood is extravasated into the alveoli and *stasi concepta putrescit*, is approved and appropriated by Niemeyer in the following words: "The blood which remained behind in the bronchi and alveoli has led to a pneumonia undergoing cheesy transformation, the retained blood and the products of inflammation afterwards breaking down, as is plainly described in the quotation from Hoffmann" (pp. 32). This appears also to be the opinion of Dr. Bäumlér, from his paper in the 'Clinical Transactions,' in which he says (vol. ii, pp. 83), quoting Niemeyer's views which he supports, "Part of the blood which is not expectorated, and which has coagulated in the smaller bronchi and air vesicles, acting by its decomposition as an irritant, and causing inflammation of the lung-tissue. This opinion appears also to be shared by Dr. Weber, although without complete acceptance of all the opinions of Niemeyer.

Although these opinions are in some measure correct, yet they do not represent the exact state of the case, these nodules not being the caseous products of a pneumonia. They differ in the following points from a pneumonic product: they are round, well-defined, and circumscribed, whereas pneumonic products are always of irregular shape, in patches, and generally shading off into the tissue near; they are tough and indurated, and show a marked tendency to separate around their circumference from the lung-tissue which surrounds them, a property which distinguishes them conclusively from pneumonic products. They are not due to the results of a hæmorrhage *in situ*, for they may be found in the lung opposite to that in which the wounded vessel or aneurism has been found,

Nor are they due to the simple gravitation of blood dribbling downwards, for they are found in the apex of the lung as well as the base. To what then do they owe their presence? The answer to this question is obtained from a consideration of the preferential regions which these nodules inhabit.

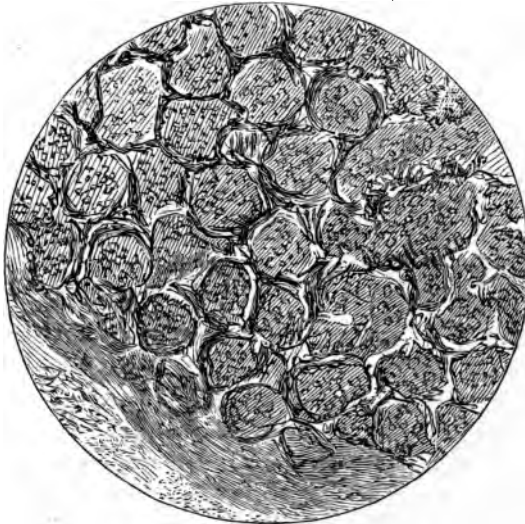
The upper lobe is sometimes a seat of these nodules, which when situated here run into each other and occasionally form rather large masses. In considering the special reasons in each case which influence the respective position of the nodules, it is necessary to consider also the condition of the lungs, inasmuch as the upper lobe is so generally affected with previous disease, either as excavated or adherent from old pleurisy. The base is another favourite locality, for they are often found close to the periphery of the diaphragmatic surface and far from being found in the most dependent part, the posterior border, they may be found limited to a region of narrow width at a place corresponding to the arched part of the diaphragm. There is also another region in which they may be found, and that is in the anterior axillary border close to the periphery, in the region of the nipple, or between the third and fifth ribs. These three regions are notably those parts of the lung in which inspiration produces the greatest expansion of the lungs, and which are most called upon in any great inspiratory effort. These nodules are sometimes found curiously isolated in the regions just mentioned, and it seems, therefore, that they owe their presence there to the inspiratory force expanding the lung in these parts, and to the force of the atmosphere driving home some substance which it meets in the bronchial tubes.

It is quite evident from the microscopical examination of sections and from the microscopical appearances that it is blood which is forced into the alveoli, and from the frequency with which both lungs (in cases of fatal hæmoptysis) show the presence of clotted blood in the tubes, the only conclusion that appears a solution of the problem is this, that in cases of profuse hæmoptysis, the welling up of the blood causes a dyspnoea, which results in forced inspira-

tory efforts; the blood which clots in the tubes is driven backwards and forced into alveoli in the apex, the base, and the axillary region; the lobules are distended with the clot, which finally loses colour and becomes hard, white, and tough, closely resembling the hard white infarcts which are found so often in the spleen, and like them may be found unaltered and inert some time after their first formation.

The microscopical examination of these nodules is a little difficult, owing to the fact that when by reason of their age they have assumed the white and tough condition, they lose a great deal of their early appearance, and the different elements become confused. But even in this stage the markings of the alveolar walls may be made out, and also the

FIG. 1.



presence within them of an opaque, homogeneous, fibrinous material.

The tissue in the surrounding region (see fig. 1) shows marked evidence of irritation, the result being similar to that which obtains in the irritation of pulmonary catarrh, with general thickening and increase of the connective tissue

elements. An examination of the neighbourhood of these nodules shows more decidedly and conclusively what has taken place. On examining the section with a lens small elevated nodules may be found, which on further investigation prove to be solitary alveoli or bronchioles plugged with blood globules firmly packed into the lumina, and in some cases distending them. The irritation which their presence excites is very marked, and occasionally leads to considerable thickening of the pleura if the nodule be situated close under the pleura, and always of the interlobular tissue which surrounds the nodules.

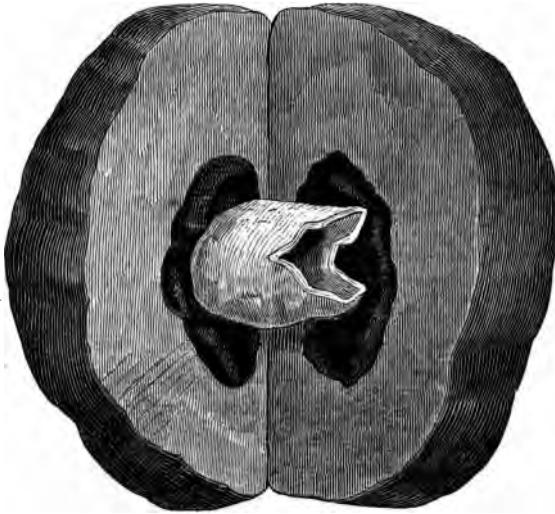
From these observations it appears that these nodules are due to the forcible impaction of clotted blood driven from a distance into the bronchi and alveoli, and that a certain amount of irritation is produced by the blood; but it must be remembered that an irritation of this kind need only result in a fibrosis of the lung, and the fibrinous mass may, and does remain often, quiescent and inert. This, however, is not always the case, and the next question is, what alterations these nodules undergo?

Allusion has already been made to the fact that they show a strong tendency to drag away from the surrounding tissue, and this especially happens when there is any strain on the respiratory tract from previous disease, or a tendency to congestive softening of the pulmonary tissue.

In two cases a remarkable condition of things resulted from this tendency (Cases 2, 3). Twice I have found the thickening envelope of the nodules lying loose in a cavity, resembling the sac of a pulmonary aneurism; in one case this free capsule was found in the fourth intercostal space and in the other quite at the base of the lung (see fig. 2). The contents of the capsule had softened at the same time and been ejected. The process of necrosis which these nodules undergo may take two forms. They may undergo a gradual process of erosion, that is, the nodule may be eaten away bit by bit, nibbled as it were by a special corrosive poison, or they may undergo a general liquefaction which involves the whole mass equally.

In the first case, that of erosion, the nodules are found

FIG. 2.



remaining *in situ*, but with bits eaten out of them corresponding to an amount of staining derived from the presence of a sanious fluid arising from sloughing of the lung tissue around them or introduced from a distant cavity (Cases 2, 3).

It is very striking to see this staining of the white nodule with the claret-coloured sanious fluid, and to note the corresponding erosion which takes place, a fact bearing strongly upon the question of the auto-infection of the lung, especially in cases of destructive sloughing of the lung tissue in which this sanious fluid is secreted often in large quantities, and of most destructive virulence.

The second process of necrosis is that of general liquefaction. When this takes place the nodule softens in the centre and gradually assumes a straw-coloured, glairy fluidity, which might be (and often is) mistaken for a purulent liquid, but the colour is too yellow and pus globules are absent (Cases

3, 5). This liquefaction is often local; for instance, it occurs at the apex (upper lobe), while the nodules in the lower lobe remain hard.

From the facts here recorded relating to the alteration of these nodules it must be admitted that hæmorrhage may result in excavation of the lung, or, in other words, they establish the possibility of a phthisis ab hæmoptoe.

2. There is another pathological condition which I understand as a condition arising from hæmorrhage *in situ*, not as a result of blood transferred from a distant locality. This is a condition which occurs less frequently than that just described (Pl. XVI); it consists of irregular blackened patches, sometimes of considerable size, as much as two inches and a half across, which may be found in the upper part of the lower lobes, or irregularly placed in the upper lobe. There appears to be no special locality for them (Cases 4, 5). They are formed of calcareous matter, loosely coherent, mingled with the black pigment of old blood, and surrounded with a defined but irregular envelope of some thickness, which is deeply pigmented with the same black granules. Occasionally the yellow colouring matter, which is often found in old blood clots, is found scattered about, indicating the original source of formation.

These patches are apparently due to hæmorrhage of some violence, producing laceration and contusion of the lung-substance, and thus leading to a secondary condition of disintegration of blood and lung-tissue, and the ultimate formation of these mortar-like patches.

In some cases, when they are found near the periphery of the lung, they assume a circular form, and ultimately, by the traction of the lung, they make their way into a bronchus and clear out from their nidus, leaving a remarkably clean and well-defined cavity. An example of this is given in Case 4, in which such a cavity had been formed, the proof of its previous condition being the pigmented state of the peripheral portion (Case 4).

With regard to the possibility of an infection of secondary tubercle following upon this hæmorrhagic condition, that

depends upon the point of view from which the subject is considered. If simply the presence of secondary tubercle is looked for, it might be admitted that it may occur as an ultimate result of such disease, but if the question be asked in this form, namely, whether the secondary tubercle arises simply from the blood products, then the answer must be that up to this time no case has presented itself in which the blood products have not undergone a secondary process, and that it requires more evidence to determine this point in a satisfactory manner.¹

The conclusions which may be deduced from a consideration of the cases which have come under my notice are these:

That in cases of severe hæmoptysis portions of the blood are driven into the alveoli, which they occupy finally in the form of fibrinous nodules, setting up some irritation in their vicinity.

That in cases of capillary hæmorrhage, with laceration of the pulmonary tissue, the resultant effect produces a calcareous mass, sometimes of considerable size.

That under special circumstances cavities may be formed by the liquefaction of the hæmorrhagic nodules in the first instance, or by the removal of the calcareous masses in the second.

That whether secondary tubercle can result from inhaled blood without the intervention of secondary processes introducing a new septic condition is a point that requires further evidence before it can be accepted.

¹ Since the above was written a fatal case of hæmoptysis in a woman has come under notice. The lung showed the presence of hæmorrhagic nodules of large size in the region of the 4th rib, at the first axillary border, and also in the anterior inferior margin, where they were found as large as peas. None were found where they are so frequently found in man, in the middle of the lowest surface of the lung.

APPENDIX OF CASES.

CASE 1.—James S—, æt. 28, seaman, admitted October 16th, 1877, under the care of Dr. Powell.

Family predisposition.—Father died of phthisis at forty years of age. Three brothers of the father died of phthisis.

Personal history.—Erysipelas of leg following an injury in 1869. Suppurating bubo in 1872.

Present illness.—Caught cold in September, 1876, which was followed by cough. Expectoration slight. In January, 1877, had an attack of hæmoptysis. Very copious for three days, chiefly in large clots. Has been unable to work for thirteen months. Much emaciation. Has lost two stone. Pain occasionally severe, with cough.

Symptoms on admission.—Cough severe at night. Expectoration stained with blood. Dyspnœa on exertion. Height 5 ft. 4½ in., weight 7 st. 12 lbs.

During November the expectoration was blood-tinged.

On the 14th November he was seized with two or three epileptic convulsions, which ended fatally on the 15th.

Synopsis.—Hereditary phthisis. Hæmoptysis, preceded by cough, in January. Death ten months afterwards.

Post-mortem appearances.—Both lungs were much congested and blood stained. The bronchial tubes were also congested and granular looking, the vessels being well marked and in a condition of hyperæmia.

Left lung.—The upper lobe was riddled with small excavations, generally of the size of a filbert, which were lined with yellow membrane and secreted yellow puriform matter. The rest of the lung was much congested. In the axillary anterior border of the lung, on a level with the root of the lung, were found one or two round curdy masses, situated close to the periphery, softening in the centre and slightly blood stained—that is, there was a slight pink stain in the centre where liquefaction was going on, and which appeared to be due to extraneous sanious matter imported into these nodules through the bronchioles. Near the root were one or two

groups of racemose tubercles. Through the rest of the lower part of the lung were disseminated some separate and irregularly formed small masses of fibrin imbedded in alveoli.

Right lung.—There was a ragged cavity of considerable size in the axillary region of the upper lobe. The rest of the lung was much congested and blood-stained. A considerable portion, viz. a band of two inches in width, of the peripheral portion of the lower lobe was studded with circular, curdy, fibrinous nodules, of a white colour, hard and unyielding, with openings of one or more bronchioles containing black pigment around these tubes. Some of them were hard and unaltered, but many were tinged with a pink sanious fluid, possibly derived from the ragged cavity above-mentioned, and gradually softening from its influence. Some of these nodules showed a distinct tendency to separate, there being an interval or chink between the circumference of the nodule and the lung tissue. There were also recent patches of inhaled blood in the same part of the lung which had not lost its red colour, and some in the middle part of the upper lobe.

In this case no aneurysm and no wounded vessel could be found, but from the general congestion and blood-staining of the pulmonary tissue and the bronchial tissue it was concluded that the hæmorrhage had probably resulted from capillary rupture.

CASE 2.—John M—, æt. 34; police constable at the House of Commons.

Family history.—Two brothers died of phthisis.

Personal history.—Had suffered from pleurisy in the left side when he was twenty years of age. In 1871 he began to suffer from winter cough. In November, 1873, he first suffered from slight hæmoptysis.

He first came under my notice as an out-patient in October, 1874, and I then found the signs of cavity situated between the third and fifth ribs, on the left side, in the axillary region. In December he had another attack of hæmor-

rhage. In July, 1875, he had another attack of hæmorrhage, which was very severe. He lost more than a pint of blood. There was another return of hæmorrhage, to the amount again of a pint, in August, and he continued under my treatment until the end of April, 1876.

He was admitted into the Hospital, under Dr. Powell, in November, 1876. He gained weight, had no recurrence of hæmorrhage, and was discharged improved on the 26th February, 1877. He continued to improve up to the 28th of August, when he had a sudden copious hæmoptysis without exertion or violent cough. This recurred again the following evening (after walking up stairs) to the amount of two pints.

He was readmitted into the hospital November 6th, 1877, and on the 18th was out walking in the grounds, when he was suddenly seized with a profuse hæmorrhage, which proved fatal.

Synopsis.—Hereditary phthisis. Old cavity of four years standing. Frequently recurring attacks of profuse hæmorrhage for two years.

Sectio cadaveris.—Body blanched and thin. Left lung very much retracted, universally and firmly adherent, so that it was removed with much difficulty. It was very much dwindled. There was a cavity at the apex, about one inch in diameter, and two and a half inches long. There was a larger cavity (2×2) situated in the axillary region between the third and fifth ribs. The rest of the lung was hard and compressed. The bronchial tubes were large and dilated; they contained some recent clots of blood.

About the centre of the pericardial surface of the lung was a cavity the size of a filbert, which was filled with coagulum of blood, and in this was a ragged aneurysmal sac which had burst into the cavity. Above and behind this were found two or three curdy oval masses three fourths of an inch in diameter, white, but pigmented with black in the centre, which were softened and eroded at the upper portions, and below were undercut, so as to separate from the surrounding pulmonary tissue.

Right lung.—Very adherent at the apex behind. It was generally much distended and expanded. No active tubercular matter was found, but some old puckered tubercle at the apex. Under the fourth intercostal space in the anterior axillary line a cavity was found; on cutting into which there appeared a round yellow bag or capsule lying loose and empty in the cavity. There was no blood nor purulent matter in the cavity. The sac would have fitted the tip of the little finger, was rough and ragged inside and out; it was evidently the remains of a curdy mass, similar to those in the other lung, which had softened, emptied itself, and separated from the surrounding lung.

At the lower peripheral margin of the lower lobe were a number of small, round, white curdy nodules, half an inch in diameter, with openings of bronchioles in their centre. Some of these were slightly stained with pink and were beginning to soften. The rest of the lower lobe of the lung was in a condition of intense congestion.

CASE 3.—H. G. C—, æt. 22, single, billiard marker, admitted first into the hospital May 8th, 1877, under the care of Dr. Powell.

Family predisposition.—Nil.

Personal history.—Diseases of childhood. In 1874 sudden hæmoptysis of bright-red blood on stooping sharply. Had noticed for some months previously a slight dry cough. Lost 2 st. last year. Slight streaky hæmoptysis, once or twice amounting to 1 oz. Weight in May, 8 st.; Nov., 6 st. 12 lbs.

May 16th.—Hæmoptysis (1 oz.) at night. Temp. 99° F.

June 14th.—Copious hæmoptysis.

28th.—Hæmoptysis, which continued off and on during his stay in the hospital.

Sept. 18th.—Hæmoptysis 5 to 6 oz.

Nov. 20th.—Hæmoptysis sudden, 13 oz.

Dec. 1st.—Hæmoptysis about 2 oz. From this time he lost strength, the pulse being rapid and very small.

He died on the 27th Dec., having been delirious a few hours before.

Synopsis.—No history of inherited disease. Hæmoptysis three years before death. Attacks of hæmorrhage frequent, and profuse in amount occasionally.

Section cadaveris (Dec. 28th, 1877).—Body emaciated; blanched.

Right lung.—Adherent behind and in front at the apex and base; adhesions firm. A large cavity full of sanious fluid occupied the whole upper lobe. The floor of the cavity was soft and flaccid, and two or three small rounded chambers were found communicating with it.

The ends of two small vessels which traversed the floor of the cavity were found corked at their mouths by a clot of blood, and which had probably been lately pervious, but were found closed a little below the ends. The lining membrane of this cavity was of a dark purple colour with a lurid congestion. Below this cavity were a number of small, well-defined, circumscribed, oval cavities, generally about the size of a small nut, that is, half an inch in diameter, closely packed together, surrounded with flaccid tissue, and floored with reddened tissue secreting sanious fluid. These cavities contained the remains of curdy nodules, which had been eroded in places by the sanious matter, and the remains of these masses remained *in situ*; pigmented bronchioles were found in the centres of these nodules. Towards the base these cavities were in some instances found to contain a glairy, yellow, puriform fluid. The lower border of this lung was firmly adherent to the diaphragm, and the pleura thickened.

Near the periphery was found a cavity as big as a walnut, which contained, lying free within it, a capsule an inch long filled with recently clotted blood, which could be shelled out of it. On making sections of this capsule it was found to be formed by interlobular tissue, which was much thickened, and inside the sac were found remains of lung tissue marked with blood and filled in the alveoli with fibrinous matter. Further section of the lung in the immediate neighbourhood of this capsule showed many little rounded masses varying

from $\frac{1}{8}$ to $\frac{3}{8}$ of an inch, with the openings of bronchioles in their centre, which were pigmented.

The glands throughout were pigmented. Some of them were washed free from blood stain, and pounded in a mortar and digested with strong nitric acid, and a distinct blue tint was obtained on the addition of ferrocyanide of potassium. At the upper part of the middle lobe below the main bronchus was found an aneurism of the pulmonary artery, as large as a pea, hanging loose by an attenuated bit of artery into the small cavity, which was of the size of a nut. On careful examination the pedicle of the aneurism was found closed by a small adherent clot of fibrine, while the aneurismal sac was full of hardening, but coloured clot.

Left lung was in a much less advanced stage of disease. It was enlarged and distended. The upper lobe was scooped out into small cavities about the size of a small filbert, which showed yellow matter close to the circumference. These cavities were formed in the same manner as those of the middle lobe of the right lung from the evacuation of the fibrinous material. The lower lobe was much congested, and of a lurid red colour. Well-defined miliary tubercle was found chiefly in the axillary region. In the middle of this axillary region were found numerous round, hard nodules, with central pigment, the size of a pea, scattered about close to the periphery. Also quite at the base, and close to the diaphragmatic surface, were found a number of hard, fibrinous nodules of a similar character.

CASE 4.—John W—, æt. 35, hospital porter, admitted Oct. 9th, under Dr. Pollock.

Hereditary predisposition.—Nil.

Personal history.—Rheumatic fever in 1870. For four months before admission had suffered from cough with hæmoptysis. Hæmorrhage of black blood on two occasions to the amount of half a pint, the last time five weeks before entering the hospital. Considerable loss of weight. Had suffered from diarrhœa.

On Nov. 20th he began to suffer from ascites, and the

urine was found to contain a large quantity of albumen, sp. gr. 1015, 50 ounces in quantity.

He continued to suffer from diarrhœa, and occasionally vomiting during the rest of his lifetime, but there was no return of hæmoptysis during the time he was under observation. He died on Dec. 28th.

Synopsis.—No hereditary predisposition. Hæmoptysis to the amount of half a pint twice; the last time being nearly four months before death.

Section cadaveris.—Dec. 22nd.—A small depressed cicatrix of old sore on the penis.

Right lung very adherent, adhesions universal and thick. The pulmonary tissue was generally very much congested, soft, and dark, containing a considerable quantity of blood. In the middle of the upper lobe and middle lobe were found two irregular patches of mortar-like matter, and surrounded by thick, black, pigmented tissue; the lower patch having a number of small, rounded, mortar-like masses, the size of mustard seed, outlying and similarly surrounded by thickened pigmented tissue. No tubercle was detected.

Left lung.—Adherent behind, with long fibrinous bands of attachment in front. Three irregular patches of mortar-like matter, similar to those in the right lung, were found occupying the lower and posterior portion of the upper lobe. A circular well-defined cavity, one inch in diameter, was found in the upper lobe. This cavity was close to the periphery, and was pigmented at its margin; the lining membrane had here contracted and become depressed; it communicated with a bronchial tube of some size (quarter of an inch). It was evidently the result of a complete clearing out of a mortar patch similar to those already described. These mortar patches were found to contain small masses of bright yellow pigment, evidently colouring matter from old blood. The lower part of the lower lobe was found full of semi-decoloured blood surrounded with blood stain and congestion.

Liver was covered with recent lymph, and was found in a very advanced amyloid condition. The peritonæum was

also covered with thick fibrinous lymph, and the kidneys and intestines were found to be amyloid. The large intestine was studded with a number of old thickened ulcers, which were probably of dysenteric origin. (Plate XV, fig. 1, and Plate XVI.)

CASE 5.—Charles B—, æt. 29, groom, admitted October 18th, 1877, under Dr. Powell.

Family predisposition.—Nil. Father rheumatic.

Personal history.—In 1864 he began to suffer from cough and hæmoptysis. He was admitted into the hospital in 1865, and left much improved. Went to Malta and India as an officer's servant until 1870. He had ague. In the winter of 1873 he was attacked with very copious hæmoptysis for several days without exertion. This returned five months before admission, but it did not recur again.

When admitted he was much emaciated; suffered severely from cough, expectoration being very copious. Towards the end of November he was attacked with epigastric pain and vomiting, and ultimately he became jaundiced, which disease proved fatal on the 9th of January, 1878.

Synopsis.—No hereditary predisposition. Copious hæmoptysis in 1873, and again eight months before death.

Sectio cadaveris.—Both lungs found to be very adherent at the upper lobe behind.

Left lung large. The upper lobe contained two cavities as big as a walnut (an inch and a half across), filled with glairy, yellow, puriform fluid. The tissue surrounding these cavities was found deeply pigmented and thickened. In the neighbourhood at the back were found some smaller, well-defined, oval cavities, filled with the same yellow matter. Below in the lower lobe were found two or three long irregular patches surrounded with pigmented tissue, and with ragged pigmented tissue in their centre, with here and there a curdy fibrinous mass adherent. These patches were about two inches long, and ran across the lung from side to side. Along the periphery of the lung, in the region of the anterior axillary portion, ran a band of pulmonary tissue half an

inch in depth, which was formed of small terminal bronchioles and alveoli choked with white, fibrinous material, apparently the result of blood inhaled into this portion. The lung-tissue at base was much congested.

Right lung.—Much the same appearance was found in the right lung, only it was less affected; the back of the apex of the upper lobe was riddled with small oval, circumscribed cavities, filled with yellow matter liquid and glairy. A similar cavity to that in the left lung was also found in this part of the lung, and irregular pigmented patches. At the upper part of the lower lobe were small rounded cavities, and a few racemose groups of tubercle tissue much congested generally.

The liver was found in an advanced condition of cirrhosis. The kidneys were small and hard.

DESCRIPTION OF PLATES XV AND XVI.

Pathological Traces of Pulmonary Hæmorrhage (Reg. E. Thompson, M.D.).

PLATE XV.

FIG. 1.—Inhaled blood: early condition showing blood clots losing colour; base of left lung. Case 4.

FIGS. 2, 3.—Inhaled blood: late condition; base of lung. Case 3.

PLATE XVI.

Mortar-like patches, result of old hæmorrhage; upper part of left lung. Case 4.

Fig. 1.

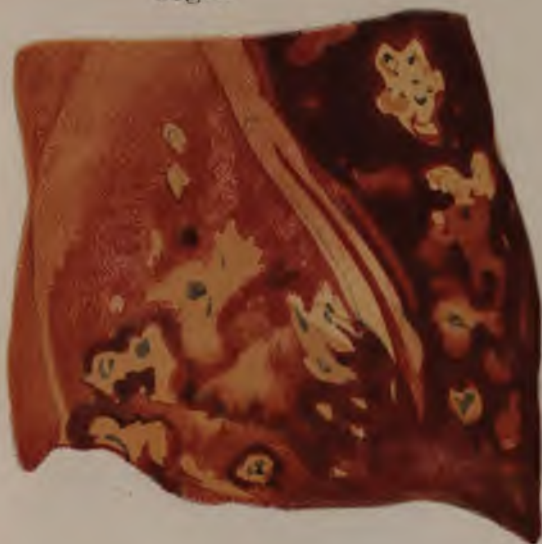
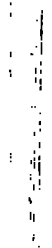
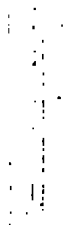
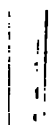


Fig. 2.



Fig. 3.







11

11

NOTES
ON THE
SPIRILLUM FEVER OF BOMBAY, 1877.

BY
H. VANDYKE CARTER, M.D. LOND.,
H.I.M. INDIAN ARMY.

COMMUNICATED BY JOHN HARLEY, M.D.

(Received February 19th—Read May 28th, 1878.)

ALTHOUGH the town of Bombay is not situated within the area in Western India lately the seat of scarcity and famine, yet it has suffered in an indirect and, so far as appears, a peculiar manner; for the crowds of indigent peasantry, flocking thither, brought in their train not only destitution and overcrowding, but also a contagious fever, which soon spread to the resident population immediately in contact with those famine-stricken people, and became the cause of death to many persons.

That this fever (which after a year or more, yet lingers amongst the poorer classes) is identical with the "hunger-pest," "famine- or 'relapsing-' fever," or the so-called "recurrent-typhus" of Europe, I hold to be now fully demonstrated; and this fact I regard as one of great significance in Indian pathology. The demonstration just referred to has resulted from the combined use of the clinical thermo-

meter and microscope; and I am not aware of any data invalidating its force. Assuredly, the negative circumstance of the same fever not having been detected in other parts of the Bombay Presidency, or in the famine districts of Madras, can in no way detract from the weight of positive proof acquired in the western capital. Such proof is as follows:—

1. The rather abrupt appearance of a malady evidently connected with unusual and excessive want, fatigue and overcrowding; its spread with the increase, and subsidence with abatement of these hygienic defects. 2. The remarkable resemblance in clinical characters of this new malady with the relapsing fever of Europe, a resemblance so close as of itself to be decisive of actual identity. 3. As a special feature, the invariable presence in the blood, during pyrexia, of a minute parasite which, so far as known, is peculiar to one form of fever only, viz. the relapsing.

Thus there are combined the natural history of an epidemic, the symptoms of a fever and the pathology of blood-contamination; each of these fundamental features displaying special characters pointing to but one conclusion.

And almost equally significant with the above, as regards diagnosis, is the observation that this new fever raged most at that season of the year when malaria least of all abounds. I should add, too, that antiperiodic remedies, such as quinine, had no influence in checking recurrence of the febrile paroxysms.

Respecting the first or natural history proof, just offered, I have to observe that its force depends upon the contemporaneous outbreak of the epidemic and the excessive influx of famine-stricken people; it is well known, too, that as the pressure of immigration augmented, the 'fever' death-rate rose; and, lastly, it can also be shown that contemporaneously with diminished influx, and with active deportation of many families to relief centres in the interior, this comparative fever mortality began to decline; though had it been due to malarious influence alone, the reverse would have been the case. Data in support of each of these statements, must be deferred, but in what is now submitted there

is nothing that is inconsistent, and much that is in accordance with accepted inferences derived from European experience. And with reference to the view of the Bombay epidemic being essentially connected with defective sanitation, I would remark, first, that there is no evidence of contemporaneous increase of mal-sanitation, and next that relapsing fever is not one of the diseases usually attributed to filth.

The evidence upon which most stress will be laid, is, however, that included under headings two and three, which treat of symptoms and pathology equally characteristic and conforming; this kind of proof is already available.

Before proceeding to details, I would first state the reasons why the term 'Spirillum-fever' has been chosen. Here, I am aware, the designating word is not quite correct (the Bacterium genus being "Spirochæte"), but it is preferred for euphony's sake and is hardly liable to mislead. The fever itself does not affect exclusively the hunger-stricken, and is neither limited to, nor the constant attendant of, famine times or places; it by no means invariably relapses; and it is commonly viewed as distinct from 'typhus;' hence the desirability of inventing a more suitable name, which if not adopted in Europe, at least might displace in India that of "remittent fever" hitherto employed.

As another prefatory remark, I would add that previous to last year I had no personal knowledge of the so-called relapsing fever; and that all now submitted is the result of considerable individual effort, a fuller expansion of my notes being necessarily reserved. It may be hoped, too, that the present enquiry will be supplemented by others made in India, where, it is right to state, the correct diagnosis of the new Bombay fever was made independently by more than one observer. I have much pleasure in acknowledging here the valuable co-operation of my two junior colleagues at the Jamsetjee Jejeebhoy Hospital—Mr. Succarani Arjoon and Dr. Anna Moreswar K,—who proved themselves to be expert microscopists.

Clinical notes; introductory and general.—Information

was collected, 1st. in the camp of refuge for fever patients opened last April (1877) and closed after too brief an interval; notes of forty-eight cases hence, several being quite typical, are now before me. 2nd. In the wards of the Jamsetjee Jejeebhoy Hospital, where most instances of the spirillum fever and contemporary pyrexias were seen, a sum of about 700 cases having been examined; and 3rd. At the Goculdass Tejpal Hospital, also under my charge, where the beginning and later pursuit of the enquiry have been conducted: at least 100 cases of fever were hence derived.

Analysis of these data shows that we have recorded upwards of 850 examples of the spirillum-fever (C); about the same number of "fevers" mostly remittents and ague, in which the blood-parasite was sought for during the presence of pyrexia, but not found (B); and lastly, a considerable number of instances of mild and transitory febrile attacks, together with sequelæ of earlier illness, in which microscopic scrutiny of the blood was not attempted (Class A). As far as was practicable I examined every case of marked "fever," making no other selection than that of decided pyrexia at some period of the illness; and the enquiry extended from April to December, 1877.

The above statement shows that upwards of one-third of all hospital—and hence, perhaps, all, ordinary—cases of "fever" were demonstrably of the so-called relapsing type (C); and large as this proportion seems, it doubtless falls short of the truth, because there are numerous instances of fever amongst the classes marked A and B, which presented a clinical history and symptoms altogether accordant with the same type and not accordant with malarious forms. I have little hesitation, therefore, in assuming that the larger part of the late serious epidemic in Bombay, was due to the presence of the so-called famine fever.

The facts just summarised have also convinced me that both "remittents" and "intermittents," in all their usual shapes, are to be distinguished by the invariable absence of blood-spirillum, once, indeed, I found a minute and active organism in the blood of a patient effected with fever seem-

ingly intermittent in type; but in general I conclude that the spirillum must be regarded as distinctive of one clinical pyrexial form. This being affirmed, I am free to remark that, previously to late Indian experience, I had but a very imperfect conception of the multiform phases and degrees which the spirillum fever may assume, for about 25 per cent. of instances could properly be termed "irregular;" and, obviously, such experience, while in accordance with late observations made in Europe, is valuable, especially to practitioners in tropical countries where fevers termed "malarious" (with their equal variety of phase) do commonly abound. European writers have briefly but clearly commented upon the similarity of symptoms between isolated, secondary, and later paroxysms of relapsing fever, and intermittent or aguish attacks; and some have owned the impossibility of distinguishing between the two series from ordinary clinical observation. Quite independently, I had myself arrived at a similar conclusion in Bombay, where it was not at all uncommon to witness a single, or even repeated, one-day febrile paroxysm, which stood in the place of a relapse, but which did not present the blood-spirillum; and on the other hand, we noticed brief and isolated paroxysms or relapses, seemingly identical with the above, during which the spirillum was readily found. In explanation of this seeming discordancy, we may, as regards the negative results, suppose either that the blood was imperfectly examined, or that ague had, in fact, complicated the case. Experienced observers in Europe would doubtless adopt the first supposition, and not without reason; but in India we hold that a malarious taint is so widely diffused that an aguish attack may be excited by almost any kind of constitutional disturbance, and hence, by that of spirillum fever. The chief difficulty in adopting this view is the fact of the non-spirillar relapse in question commonly being true to time, and so giving rise to the impression that it stands in place of a veritable relapse. This interesting topic can be best discussed with the aid of charts, which at present I am unable to transmit, and hence must be content with the statement

that, on the whole, we have not in India secured valid evidence of intermittents (which I regard as the type of all periodic fevers) being, either partially or wholly, substitutory of the spirillar-paroxysms. That ague may precede, accompany, or follow the spirillum-fever, and that each of these types may possibly predispose to the other, I should not hesitate to admit; but until we find in the blood of individuals affected with ague, an organism comparable to the spirochæte, it would be premature to assert a generalisation plausible only in theory. I have met, indeed, with an instance which seems to support the view of an essential connection between the spirillum-fever and intermittents, and which is certainly remarkable; but as it is also as yet single and seemingly exceptional, this subject may stand over for the present.

There exist, however, more important general resemblances. So far as I know, single, primary, and more pronounced attacks of the spirillum-fever, when free from complications, never assume the character of an intermittent, but they may often be compared with paroxysms, more or less restricted, of the "remittent" type. The pyrexia in a typical example of spirillum-fever may, indeed, be "continuous" in character; but it is, at least as often, distinctly "remittent," or both remittent and intermittent, so varying is its intensity in some instances. Were it possible to conceive of brief remittents which periodically relapse (I have never seen anything of the kind) then it might be difficult, at sight, to distinguish them from the true spirillum-fever. The use of the microscope would, however, dispel the illusion, which might also be dissipated by careful attention to other symptoms besides that of temperature, and to the natural history of the complaint.

Again, the interval or apyrexial periods of spirillum-fever may be so filled up, as it were, with daily changes of temperature as to render it by no means easy to make out the date of true relapse or even its cessation; nor is this filling up necessarily due to local inflammation, at least there may be no appreciable signs of the latter, and one seems obliged to

assume a previous malarious taint which has become roused into action, as it were, and so complicates the newer specific fever. Repeated scrutiny of the blood will serve to establish the times of appearance and disappearance of the parasite; but too few of these remarkable cases of prolonged fever, attended with single or recurrent development of the spirillum, have been seen to enable me to decide upon other diagnostic signs; only, as regards the late epidemic in Bombay, I acquired the impression that it was "typhus" rather than a "remittent," which is apt to complicate the spirillum-disease. In practice, the latter was found to maintain its periodicity, high temperature, and blood-contamination under all conditions, *i.e.* whether complications existed or not; and this feature serves to indicate its true special character. The most apparent symptom, namely, high temperature, is not, however, its most constant mark, for I have known the complicated specific paroxysm to be attended with, at first, rather a diminution of fever-heat, with a greater variation in its course, and at its close with a decided fall of temperature, the spirillum then ceasing to appear in the blood notwithstanding the prompt resumption of high fever, and reappearing only with a relapse. What is known as "the typhoid state" was, under various conditions including those of remittent fever, sufficiently familiar during the epoch under review; and when fever cases were first seen in this state practical difficulties were found in their diagnosis. Under such circumstances attention was given to the previous history, and search made for special signs or symptoms, local and general: microscopic examination of the blood never being omitted, and furnishing results which, even if negative, were commonly useful. Detection of the spirillum always, in my mind, inclined to a more favourable prognosis than might otherwise have been made, due watch at the same time being maintained for complications, and for that sudden collapse which often attends the disappearance of the parasite and the inauguration of convalescence. I do not regard the effect of quinine in these advanced cases as by any means necessarily diagnostic; the drug was freely

administered, but, as before intimated, it has no specific action in the spirillum-fever. The difficulty of distinguishing the latter complaint from fever symptomatic of local inflammation as well as from remittents attended with complications, may, when the use of the microscope is neglected, lead to fresh confusion which is not removed by an autopsy. Thus, for example, I have known "pneumonia" to be entered as the name of the disease, when it is morally certain that the case was one of specific fever early complicated with lung inflammation.

Illustration of these remarks (which might be extended) could be afforded only by citing individual cases; I therefore simply add that considering its multiform phases, (only newly learnt by us) it is hardly a wonder that the late epidemic has been prominently regarded as belonging to the malarious remittents; yet I am bound to add that this identification is not held by those practically acquainted with the disease, and it is much to be regretted that "remittent fever" remains as the official designation of a malady wholly different in origin and character. Were it only, indeed, because of the almost infinite confusion which lurks under this nosological heading, being thus rendered more confounding, it would be well not to delay a much needed remedy, but rather begin with the elimination of so marked and important a complaint as that of "relapsing-fever;" and then other just discriminations would follow—those of "leprous" and "elephantoid" fever being already practically acknowledged. I may here note that during an attack of obscure elephantoid fever the blood *filaria* was to be found, but on subsidence of the pyrexia I could no longer see it; here was evident an analogy with the spirillum fever.

A final remark will complete this cursory review of fevers simulating or complicating the new Bombay form; I refer to the continued or typhus type, of which there have been seen some unmistakeable illustrations. And first as regards certain remarkable connecting links, I have to record that both at the height and towards the close of the epidemic (my experience points oftenest to the latter) cases occurred

which had all the characters—the blood spirillum included—of the so-called “typhus icterodes” or “bilious typhus,” in a most pronounced degree; instances are known also of the equally fatal type “hæmorrhagic typhus;” and in short our late experience here has been in striking accord with that acquired in great European epidemics. Many cases, too, of single or abortive spirillum-fever—not to allude further to severer examples—have presented an amount of constitutional depression which, in my estimation, would almost justify the designation of “typhus,” notwithstanding the absence, or seeming absence, of some cutaneous hæmorrhage or mottling. As a rule, however, unless there be seen indications of the characteristic temperature changes in some part of their course, it would be futile to search for the spirillum amongst instances of high and prolonged pyrexia (the only exception I have met with, was not seen until at a late stage); and when a fever can be said to have assumed other specific characters, *e.g.*, such as “typhus,” the parasite in question will, most probably, not be found.

This remark holds good of enteric or typhoid fever (in which with one very doubtful exception, the spirillum was never found), and I may add of small-pox and varicella, and numerous symptomatic fevers whether isolated or occurring as complications.

From what has now been advanced it will be evident that the subject in hand is by no means so simple as might at first sight appear, and in attempting to unravel this intricacy it would, perhaps, be best to recognise the entire pyrexial condition itself as a common character of several affections, each of which has as well its special features in mode of origin, local lesion, course, or termination. This view would imply a certain wider bond between all fevers, whether idiopathic or symptomatic, and a closer one between those commonly termed “specific,” wherefrom one might hope for general illumination by particular inquiry. Certainly, the characters of the spirillum-fever are, as it seems to me, very comprehensive, combining, as regards malarious fever, the intermittent (through its marked periodic tendency) and the

remittent (in its sustained paroxysms), and occupying an intermediate position also—through its connection with typhus—with the continued type of fevers; characteristic, too, is the remarkable clearness with which the attendant, if not causative, parasite is displayed in the blood, and hence, with unusual confidence, we may anticipate substantial benefits to pathological science from the thorough study of this particular form of "fever." Amongst other topics of inquiry I would indicate the desirability of establishing the "type" of spirillum fever, or, in other words, of ascertaining the conditions of relapse; so varied, too, are the phases of this most interesting malady that I would ask, ought not the fever to be regarded, like those due to malaria, as embracing a "class" of ailments? And, lastly, there is the question of its true affinities.

Symptoms, mortality, and pathology.—As virtually a new disease in Bombay, interest naturally attached to every part of the clinical history of the spirillum-fever; and in practice interest ripens into importance, for the fuller our knowledge of details, the better able we shall be to recognise and adjust such anomalous and obscurer symptoms as are often presented by patients who are first seen at intermediate or later stages of their complaint. Usually, however, the sick came in not with sequelæ or complications, but towards the close of the first paroxysm or interval, and then the relapse alone was seen in full. The entire series of phenomena was visible only in the case of hospital servants and apprentices, and of patients freshly attacked in the wards. My own case, too, was a fair example of moderate severity, and of all these complete instances I may say that their symptoms entirely correspond to the classical descriptions of European authors, the variations therefrom being no greater than the inter-variations found in Europe itself. Thus, the attack was sudden and unexpected, the introductory chills not very marked, the fever at once high and then continuous or but imperfectly remitting; there were frontal headache, giddiness, and aching pains; much weakness, thirst, and nausea; sleeplessness, restlessness, and not seldom towards the end of

the paroxysm some delirium at night ; constipation and yellow tinge in the conjunctivæ ; abdominal fulness and epigastric uneasiness ; the spleen and liver also participating : when at its height, about the seventh or eighth day, the general distress abruptly came to an end, with critical sweating and considerable fall of temperature—in my own case from 103° to 96° Fahr., but I have witnessed a descent of 10° and 11° within a few hours, attended with most pronounced collapse. The apyretic interval now begun allows of complete rallying, if not convalescence ; thus, in four or five days I was able to leave my room and take a short walk, but on the eighth day a relapse came on, and shortly the temperature rose to 105° , falling later to 95.8° ; with the pyrexia again came headache, pains, sleeplessness, and wandering, such weakness that actual syncope was induced by the upright posture, an enlarged spleen and vomiting, with diarrhœa ; the feet were puffy. In three days the crisis occurred, with a very sharp rise immediately after, and next day a temperature oscillating about the normal. An imperfect second relapse was noted and convalescence was protracted. The blood-spirillum abounded during the first paroxysm (as, even during the fever, I was able to make out), and this fact confirmed the diagnosis which rather suddenly flashed upon me with the recollection that I had a few days before scratched my finger at the autopsy of a patient affected with this fever. Inoculation, however, is by no means necessary to infection, and my state of health, weakened by long exertion, may have predisposed the system to disease at the end of six months' continuous exposure.

Diverging from these plain instances of fever with one relapse, we find, on the one hand, attacks in which no relapse occurs—and these, more frequent of late, have amounted to about 25 per cent. of milder or recovered cases ; if, however, instances fatal during the first paroxysm be added, these, no fewer than 40 per cent. of attacks of the spirillum-fever in Bombay, did not show a relapse. On the other hand, there may occur two, three, or four relapses ; and long subsequently

a renewed attack of the fever. Taken on a series of 177 cases not fatal (the addition of deaths would make no material difference) 57 per cent. of all instances were known to relapse once, 6 per cent. twice, 5 per cent. thrice, and 2 per cent. four times. In my estimation these figures alone would dispel the idea of comparing the new Bombay fever to any form of "remittents;" and further to show its identity with European famine-fever, I may observe that the mean duration of the first paroxysm was seven to eight days, the range being four to ten days; when a relapse took place it was after an interval of a week or so, and it lasted four to five days, with a range of one to seven days. No difference was noted between the abortive paroxysm and that followed by a relapse, and no fixed relation was observed between the severity of first and second paroxysms.

Repeated attacks of spirillum-fever in the same individual, after an interval of a few weeks or months, were well known in Bombay, but it was impossible to say whether or not they were due to a fresh infection.

Briefly adverting to individual symptoms, I believe there is no feature peculiar to Indian experience. Prodromata seem to be rare, and they were not mentioned in an instance where I found the spirillum about an hour after chills and the beginning of pyrexia; in my own case they were not noticed, and it may be said that relapses in general are seldom preceded by admonitory symptoms. These facts are worth attention, because one would anticipate some systemic disturbance with onset of the parasite, in its earlier stages of development.

The dark flushed, dusky and helpless physiognomy of fever patients became very familiar to us; a peculiar faint odour from the body was at times perceptible; in my wards a pink-coloured eruption (at first resembling the typhoid) which might become permanent, was not uncommon and petechiæ were present in severe cases; also sudamina and occasionally herpes and bullæ; the pulse and temperature ranges were absolutely identical with European standards; the weakened systole was common; œdema of the feet frequent; and

also pulmonary congestion. The tongue was dry and brown oftener than is indicated in European experience; it might remain so during the first days of a pyretic interval, coexisting probably with delirium and other typhus symptoms. Epigastric tenderness was not necessarily connected with vomiting, nor hepatic enlargement and tenderness with jaundice; the spleen was seldom tender on pressure, but I have seen the reverse when after death there was no "splenitis;" this organ is subject to rapid changes of volume and with acute enlargement vomiting has been noted. Both hepatic and splenic implication were most frequent and decided during the relapses. Jaundice was commoner here than it usually is in Europe; and the same remark applies to delirium, which was often acute, and marked by a suicidal tendency (two of my patients destroyed themselves by jumping out of window, within a few days of each other; yet in neither case was the delirium turbulent); deafness was a not infrequent sequela, but ophthalmitis very rare; and numbness and weakness of the lower limbs sometimes long persisted; mania and mental fatuity have seemed to follow an attack of spirillum fever. I have but few notes on changes in the urine; albumen was seldom found during the pyrexia.

Abortion in pregnant women occurs here just as in Europe and was early witnessed. The ravenous appetite has been often noted.

Even the complications (pulmonary oftenest and buboes, next hepatitis and bowel complaints) have proved to be similar to those met with in the West; and the same remark applies to the sequelæ, debility, emaciation, abscess, rheumatoid, paralytic and other nervous symptoms. Parotitis may be late or early. Other details might be furnished, but already, and from what follows, the true nature of the Bombay fever will become sufficiently evident.

Mortality.—As derived from hospital statistics, the death-rate of the Eastern epidemic will appear excessive; it has, however, varied considerably: thus in May (the worst month observed) it was 20 per cent. of admissions, but only 10·5

per cent. in September, the mean of five months being 18·7 per cent. All my notes are not yet arranged, but allowing for contingencies, I should estimate the average death-rate at 10 per cent. of those attacked; and if this ratio be applied to the mortality returns from the whole town, it will be seen that the amount of "sickness" has been immense indeed. I estimate the death-rate of ordinary fevers (malarious chiefly) seen during the period under review, at more than double; and that of severe remittents above treble, the rate of mortality just mentioned. Age and sex have not seemed to exercise much influence on this rate, but the more practical point of fatality according to stage of disease is interesting; thus, analysing between thirty and forty deaths from demonstrated spirillum fever, I find as follows:—about 50 per cent. happened within the first week or during the first paroxysm of the disease; 25 per cent. more, during the first interval succeeding; about 15 per cent. occurred during the first relapse, or in the following interval; and the remaining deaths took place at subsequent periods of a few weeks, from dysentery, parotitis, or other sequelæ. The fact that three-fourths of all deaths happen during the first paroxysm, or just afterwards, is sufficient evidence of the severity of this disease as witnessed in hospital practice, and a large proportion of sufferers owe their death directly to the fever itself. Speaking generally, the epidemic became less fatal with its gradual subsidence; but latterly there have appeared a series of fatal cases, which, in character and severity, show a tendency to the typhus-type.

Anatomical lesions.—Still of necessity confining these notes to a summary, I find no lesion (excepting that of the blood) which is absolutely peculiar to the spirillum-fever, nor is there any ordinary morbid alteration which is limited to one particular stage of the disease, or to any single organ. Some change has been noted in all the thirty-seven autopsies as yet analysed, and much the commonest was enlargement, with or without congestion, of the spleen and liver, yet how far this is due to the spirillum-disease might be questioned in India, where malaria reigns, were it not the fact that in

Europe the same thing has been found. Taking the organs of the body in succession, the alterations noted were as follows :—

Brain : in 20 autopsies congested 8 times, showing some opacity of the arachnoid 3 times, and meningeal hæmorrhage 1 ; serous effusion 2 : no evident change 4 times, including two instances of death during the first paroxysm. In a smaller series of autopsies not here included, meningeal and diffused hæmorrhages have been found : the *spinal cord* was examined and was unchanged. *Lungs* : noted as healthy 13 times in 36 autopsies, congested 10, inflamed 6, collapsed 5 times, the seat of hæmorrhage 1 : pleurisy once : the pneumonias were chiefly in the first interval of the disease, when, too, collapse of the lung was most frequent : congestion occurred during the pyrexia mostly. *Heart* : seldom diseased, though clots in the cavities, especially of the right side, or both auricles, were noted 25 times in 36 examinations ; it was fatty in aspect 4 times. *Liver* : enlarged, with or without congestion, 17 times in 37 autopsies ; regarded as fatty 3 times ; as healthy 7 times, including 4 cases dying during the first paroxysm or first interval. *Spleen* : enlarged 19 times in 37 examinations, and sometimes very considerably ; there were found fibrinous infarcts 8 times (almost exclusively in subjects dying early in the disease), and 3 examples of softening of the spleen in 15 autopsies made after death during the first paroxysm. *Kidneys* : were noted as healthy-looking 10 times, congested 9, pale 6, large 5, fatty (?) 4 times, granular (?) 1, and with infarcts 1, in all 36 autopsies. *Intestines* : 17 times healthy in 27 examinations ; small intestines congested 5 times, hæmorrhage 1 ; ulcers in the large intestine 4 times.

The post-mortem examinations conducted at the Goculdas T. Hospital, do not differ in results from the above. Frequent microscopic investigations of the principal organs were made, but my short-hand notes are as yet undeciphered ; I may remark, however, that granular and fatty degeneration (sometimes acute in character) of the hepatic and renal gland-cells was commonly found ; fatty degeneration of the

heart-muscle does not occur nearly so often, and it may be quite absent when during life the pulse was very feeble. When necropsic results are arranged according to stage of the disease, the proper plan to follow, I find, as before intimated, no invariable connection between lesion, organ and progress of illness; nor does the presence of the spirillum in the blood, or its bequeathments, inevitably entail such local changes, *e. g.* of a physical character, as might be anticipated; hence, to all appearance, death may ensue directly from the fever; but further observations are needed with reference to the dynamic, physical and chemical changes causing death.

Etiology.—The predisposing causes of the spirillum fever vary in importance; whether sex is concerned has not been made out here, yet I think the mother of a family was affected first oftener than after her children; age has no peculiar influence, the most common periods of life in my lists being from fifteen to thirty years; infants at the breast and the oldest people may suffer. In marked contrast to malarious fevers, and especially the remittent, the season of the year has not seemed to influence the progress of this new epidemic, or if an influence were exerted, it was in a precisely opposite direction. Thus, as is well known, malaria abounds least in the hot season when the soil is parched and vegetation dried up, yet it was in May that the spirillum fever reached its maximum of intensity; and, on the other hand, after the heavy rains, when malarious fevers become common and severe, the same fever had already far advanced in its uninterrupted decline and was not rekindled. As an epidemic it began in the cold weather (December to February) 1876-77, increased rapidly until June when the rains begin, and subsequently declined in a gradual manner until the close of 1877, or the date of my last notes—being then by no means extinct. In India, such facts as these are significant.

As to occupation, none is spared; immigrant labourers and their families (chiefly), weavers, cab-drivers, railway servants, petty shopkeepers, mill-hands, school-boys, domestic ser-

vants, hospital servants and attendants, students and surgeons; these and many others are in our lists. In entire accordance with this, the fever was strictly localised in the poorest and most overcrowded quarters of the town; and I am assured that very few cases, indeed, occurred amongst the better housed and better fed of the native population. The European community was virtually exempt. If these data be accurate (and I believe them to be so) the nature of the Bombay fever should be no longer doubtful; and I know of no facts contradicting the inference that we had here to deal with an epidemic essentially due to destitution and overcrowding. In making this statement I do not forget that the worst fed and housed are always the greatest sufferers from disease, including, in the tropics, malarious fevers; nor is it denied that the famine-stricken immigrants must have at least equally suffered from like maladies.

Adverting to the more immediate causes of the spirillum fever I would look for them in want and contagion, that is to say, to the conditions which alone have been found influential in Europe. One of the earliest cases I saw was that of a destitute lad who, while tramping to the Presidency town, from the famine district of Poona, was seized with fever in the upland country, and was barely able to crawl to hospital immediately on his arrival in Bombay; it was then the seventh day of the fever according to his plain story; the symptoms were typical and I found the blood to contain abundant parasitic elements; next day the usual crisis occurred, the spirillum disappeared and there was no relapse. Such instances were numerous, and they seem to show the likelihood of the spirillum fever originating *de novo* as the result of want, toil and exposure; there remains, however, the possibility of this lad and others like him having somewhere, and at some recent time, been exposed to infection whether of persons or things; and that this, or an equally potent third factor, is concerned, appears from the following narrative. A father and young son tramping from the same district up-country to Bombay, at a short interval of time afterwards, were also seized with fever on the journey

and came at once to hospital, where the symptoms proved to be quite typical of quotidian ague; the blood of both individuals was repeatedly examined without any trace of the spirillum being seen. I am aware that even precise instances of this kind have but a limited value in debate, yet they are such as are met with in practice and upon their interpretation opinion much depends.

If the originating influence of want be admitted, still it will be evident that not all, or nearly all, cases of spirillum fever can thus have arisen; and in Bombay as elsewhere almost every circumstance in the natural history of the disease, which is usually regarded as evidence of contagion, has been abundantly observed; thus, the fever rapidly became epidemic at a non-malarious season of the year, and it distinctly spread from the destitute immigrant to his family, host and neighbours, no other interpretation being possible of many instances coming before us at hospital, where nothing was more common than the admission of whole families down with this fever, whose members had been successively attacked, and their houses were not seldom found to be foci of the disease, in one instance eighteen sick persons being traced as coming from a single dwelling. Of the examples—not a few—of patients in good bodily condition suffering severely, it can only be said that in all of them there had existed the possibility—in several the extreme probability—of direct contagion; amongst such instances of spirillum fever in the well fed and fairly housed, were those occurring in the hospitals themselves amongst servants, ward attendants, and medical subordinates. The two native gentlemen and myself who undertook the task of investigating the new disease did not all escape, for Dr. Anna, M.K., had a severe attack of the fever, and I also at a later period. Lastly, it happened that several patients admitted for other ailments became, after a period of time surpassing the longest agreed upon as that of the incubation of relapsing fever, themselves attacked, and of all these it may be said that they had been exposed to contagion.

No suitable opportunity has occurred of measuring pre-

cisely the incubation period of the spirillum fever ; but I have always regarded it as corresponding to the apyretic interval which separates one febrile paroxysm from the next.

There has been no evidence of the new fever arising from malaria, and there is none (that I know) of malarious fevers being due solely to want and overcrowding.

That the spread by contagion was not more glaringly apparent may be due to the influence of a tropical climate, for the cattle disease did not spread in Bombay with the celerity that it does in England.

The spirillum fever of Bombay was not communicated to rabbits and pigeons by inoculation with infected human blood or by injection of the same into the veins.

Lastly, that the greatest misery, densest overcrowding, and a high mortality prevailed for months in the low quarters of Bombay city, is known to many residents. I myself found in inspecting certain of these localities, and other official reports bear ample testimony to the fact, that by the immigration of thousands of families, starved at home and driven to seek work for food in the Presidency town, the usual influx of labourers during the cold season was augmented to such a degree, that existing dwellings (no new ones were built) became over filled, food scarce, and the death-rate from "fever" so high, as sometimes to exceed the total death-rate from all causes in previous years. The authorities were at last moved to act, and with diminished overcrowding, the epidemic began *pari passu* to subside.

Thus I should interpret our late experience, and reverting to the past, it is interesting to note that during the years 1864-5, an almost similar state of things existed in Bombay as a consequence of marvellous commercial prosperity (not famine). At that time overcrowding became excessive, and a febrile epidemic arose (it was nothing less), which by good authority was referred to the typhus type ; and then as now several of the attendants and pupils at the large Native General Hospital were attacked with fever, which proved largely fatal. At this distance of time I cannot ascertain if

there then coexisted fever of the relapsing type, but that this latter is no new thing in Bombay, would appear from a cursory inquiry I have made into hospital records of late years, which show that occasionally there have appeared cases of fever amongst the poorer classes displaying the general characters of the spirillum disease, and one might ask if it be not from such sporadic examples that the idea of a relapsing remittent has been derived. It is, perhaps, as much by tradition as by observation, that here, nearly all idiopathic fevers are still attributed to the influence of a pervading miasm; but even the more conservative amongst us have had to allow that veritable enteric or typhoid fever is to be seen in Bombay, now the true famine fever has to be admitted, and I should myself add typhus to the list of great pyrexias rescued from the oblivion underlying the term "remittent fever" as commonly employed.

2. *The spirillum and other blood-elements.*—My observations were begun in April last (1877) with the microscopic examination of blood taken by means of a clean needle from the washed finger of a fever patient in the Camp of Refuge; the glasses used were carefully cleansed, and an excellent $\frac{1}{16}$ th inch immersion lens by Powell was employed: subsequent proceedings were conducted in the same manner, the blood being examined immediately on withdrawal, and afterwards if needed. Experiments were made by submitting the specimens to an elevated temperature, and all appearances seen were noted in order—blood-plasma, fibrine-bands, and rate of coagulation, red corpuscles, white globules, protoplasmic bodies, granule-cells large and small, free granules, rods or filaments, and finally the spirillum. The times of examination were 9—10 a.m., and 2—5 p.m., which correspond to the intervals between meals; and the seasons were the hot weather (April—June), the rains (June—September), October and November (when malarious fevers become common), and part of December (cold season). The subjects examined were of all nationalities and castes, no differences among them being perceptible; each patient was, as a rule, examined on admission, and on several subsequent

occasions, but from want of time I had sometimes to be content with single observations when these were of a positive character, the diagnosis being then regarded as sure, and experience invariably justified the assumption. Healthy blood was from time to time submitted to scrutiny, in order to rectify possible errors; and the sweat, saliva, secretion of blisters and sores, the urine, &c. of fever-patients were also examined at intervals. A bare summary of results can be offered in this place.

1. The blood-plasma is clear, clouded or granular. Almost invariably in the pyrexial stage, where spirilla abound, it is remarkably clear, and to the experienced eye at once characteristic: in pure remittents also it is pellucid, whilst in temporary pyrexias and symptomatic fevers it is apt to be much clouded. When free protoplasm abounds, the plasma is usually thick and even granular in aspect: it is clouded often at the beginning of the interval, *i. e.* immediately after the paroxysm, in the spirillum fever, but no absolute rule obtains in this matter. The plasma speedily separates into fibrine and serum.

2. The fibrine-bands during the pyrexia are usually very distinct, and brilliant almost as crystals; they entangle in their meshes, and so seem to stimulate, the wriggling parasite which endeavours (as it seems) to escape. The bands are thicker than the spirillum, straight and quiescent; their visibility is partly dependent on the translucency of the serum, and they have also a connection with free protoplasmic matter.

3. White globules. I do not regard the superabundance of these bodies at the close of and after the febrile paroxysm as very significant, because habitual leukæmia may be present also; and the excess is seldom marked.

4. Red corpuscles. These are clear and perfect in typical spirillum fever, and frequently their meshes become, after coagulation, the refuge of the spirillum whose movements are then imparted to the corpuscles, and so produce a twitching or twirling, which may for a time be the only sign of the presence of the parasite. Sometimes there is seen a sticking

together, as if some affinity existed between the corpuscle and spirillum; and the latter will drag about the former causing it to alter its shape; this adhesion may persist after death and is probably mechanical.

5. Granule cells and protoplasmic masses, may be numerous and prominent objects in the field; they include, *a*, large nucleated globular cells which may change their form, become vacuolated and undergo fatty degeneration; possibly they are derived from the spleen; *b*, large, flat cells, probably endothelial, from the inner membrane of artery, vein, or lymphatics; these possess a fixed contour and may also become charged with fat-granules; *c*, masses of protoplasm of varying size, and when large capable of assuming great variety of form; they throw out also long and slender mobile filaments, which may simulate the spirillar body. Diffused protoplasm has the form of little clumps, which are apt to aggregate in clusters of peculiar aspect.

6. Free granules are not uncommon, but probably not limited to the spirillum fever; their character is probably various, some being fatty, some albuminous, and some resisting the action of both acids and alkalies; whether or not the granules which seem to grow out into rods are perpetuating spores or germs of the parasite, is as yet undetermined.

7. Rods or filaments. Under this heading I have included various minute bodies, whose character is still uncertain; when detached they may resemble the spirillum so much as to give rise to the idea of their being an early stage of its formation, but I have sought in vain for the succeeding stages when spiral form and active motion would be imparted. They exist oftenest along with the active parasite, but in other fevers may occasionally be found; seeming to arise either from free granules or from protoplasmic extension, and in the latter case are, for a time at least, attached. A certain amount of proper movement may be noticed in them; one extremity is usually somewhat expanded and their contour may be wavy.

8. *The Spirillum (Spirochæte, Ehr.)*.—Its ordinary aspect is that of a colourless, homogeneous, and very delicate,

spirally-twisted filament, which during life is in constant motion amidst the *liquor sanguinis*; the length varies from $\frac{1}{1500}$ th inch to $\frac{1}{500}$ th inch, its breadth is immeasurably slender, or probably not more than the $\frac{1}{30000}$ th to $\frac{1}{50000}$ th inch. The spiral or cork-screw twist is regular, and best seen when the organism is quiescent or after its death, whilst during active movement (especially the lashing side movements) it becomes undone and may almost disappear, the filament meantime gaining in apparent length. The motion of the spirillum though continuous, is transiently intermittent and not constantly in the same direction; it is compounded of a rotation on the long axis—which is the most characteristic—a lateral or lashing movement, and a progression forwards or backwards; all these actions may be so rapidly performed as to puzzle the eye of the observer, who sees only swarming or shooting non-defined bodies traversing the field of the microscope. The filaments have neither head nor tail, possess a firm consistence and are highly flexible and elastic, but not contractile in the proper sense of the word; a faint yellow tinge has been noticed within them, and their visibility partly depends upon the clearness of the medium they inhabit. Not much variety in their general aspect and characters has been detected, that of dimensions being the chief. Vital movements may persist some time after death of the body (I have seen them thirty-five hours afterwards), or after removal of the blood from the living subject; they gradually cease, a brief lashing or twirl being noticed at long intervals in the midst of the countless bacteria and bacillar rods which so soon grow up, and finally the spirillum disintegrates and disappears, leaving no organic trace behind.

The number of spirillar bodies in the blood of a patient suffering from this specific fever, is simply immense, and often incalculable except by hundreds of millions; dozens may be seen in the field of the microscope at one time, troubles of the circulation being clearly enough indicated in the patient's dusky and harassed visage; on the other hand, the spirilla may be rare and to be found only after very patient

search, but it is seldom that only one is seen, and it seems probable that when present they exist in considerable numbers. Doubtless a relationship obtains between the abundance of the parasite and the intensity of the fever, but it is a contingent one; for I have observed the spirillum to be tolerably numerous during collapse (temp. 95°, F.), and at ordinary temperatures, while a rise in heat when considerable (105° to 106°) may be attended with a positive diminution in the numbers of the organisms, and is consistent with their entire disappearance. Commonly, however, pyrexia and parasite are in closest relationship, at particular times; but I have failed to verify some of the rules laid down as regulating such connection. In Bombay a rise of temperature (without complication) initiating either first paroxysm or true relapse is not invariably preceded by the appearance of the parasite in the blood, and in the rise pertaining to pseudo relapses, the spirillum will not be found at all; it is also absent during the fever symptomatic of local complication, and its advent during a continuous high temperature may be held correctly to indicate the supervention of a specific attack, which will pursue its course independently of attendant conditions. Lastly, I have met with the parasite at periodic dates, when there was absolutely no rise of temperature. Amongst the variations noted by us that of aggregation of the spirilla is prominent. I believe this may occur during life, difficult as it is to conceive how the circulation can be properly carried on while the blood contains masses much exceeding the diameter of most capillaries, and sometimes enveloping protoplasm or red corpuscles themselves. In short, the idea is inevitable that we may see here the foundation of a physical pathology, so striking are the appearances noted. The activity of the spirilla in these dense clusters is sometimes so great, as to impart a resemblance of swarming zooglœa or micrococcus colonies; and to give rise to the impression that the groups may have originated from separate centres.

A less prominent aspect is that of filaments joined together at one end or at one part of their length, whence

result many varieties of form. When adherent at one end, two or more filaments may extend almost or quite across the field, forming long vibrating threads, like the wires of a telegraph, stretching in different directions; and such threads abruptly parting, simulate multiplication by fission.

The spirilla do not always thus cluster or adhere, but they have a natural tendency to seek a liquid medium and so collect in the pools of serum left after coagulation of the blood, where they may often be found when in the solid parts, or clot, they are invisible; hence in doubtful cases, it is well to re-examine a given specimen of blood after an interval of a few hours.

Of the natural history of this remarkable parasite nothing new has been learned in Bombay. I have, indeed, ascertained, by repeated examinations, that the contents of the *thoracic duct* are always free from contamination, at times when the blood swarms with the spirillum; so that one might be led to infer the germs or spores of the organism do not enter the blood *viâ* the food or digestive canal. Such microscopic scrutiny as is practicable of the solid viscera—lungs, spleen, liver—has led to no serviceable illumination as to the source or lair of the spirillum, nor have my examinations of the blood in different localities—*e.g.* right and left side of the heart, portal and hepatic vein, splenic artery and vein—as yet led to certain results. With me, as with others, attempts to cultivate the parasite have failed; thus blood containing spirilla taken during pyrexia, and specimens taken both before and afterwards, have been kept at fever heat for hours without any notable change appearing; a very high temperature (110° F.) probably hastening the death of these organisms. It seems to me highly probable that the spirillum is developed in the blood itself, yet if this be so, it is strange that no intermediate stage of development or growth has been satisfactorily made out in the course of many hundred blood examinations. We have learned, indeed, that the bacterium may appear and disappear within the interval of a very few hours, and that its continuance during an entire febrile paroxysm of several days

duration, is most probably effected by successive generations, but whence these latter come and how they are developed are questions I own myself unable to answer.

The influence of such reagents as water, common salt and quinine in solutions of varying strength, upon the spirillum outside the body, was inquired into; and from our brief experiments it seemed that soluble quinine, much diluted, had the effect of decidedly bringing the movements of the parasites to a standstill.

As to the identity of the blood spirillum with similar filaments to be found in the saliva of fever patients, it might be difficult from appearances only to disprove this; yet I note that the spirillum (spirochæte) of the saliva is present in the apyretic interval, and, even during the pyrexia, does not vary in numbers at the same time with the blood spirillum. Further, my own saliva contained the same filaments when I was conducting these inquiries, and was in good health, excepting occasional indisposition from ordinary feverishness.

The sputum is indeed excellent breeding ground for the lower organisms, and so may the blood itself be, for in one of my fever cases, six and a half hours after death, it was found to swarm with long and mobile bacillar (?) filaments, &c.

Regarding other identifications which could throw light upon the source of the blood spirillum, my note book shows that in a tank at Bombay resorted to for drinking and washing purposes by the friends of two children who were attacked with the spirillum fever, there existed both before and after the rains, an active filamentary body which, on comparison with his plates, appeared to be Ehrenberg's *spirochæte plicatilis*. Rather contrary to my anticipation, its dimensions were so much in excess of those of the blood spirillum, that I cannot suppose the species to be the same, and we have no evidence that the spirillum fever is due to contaminated water supply.

DESCRIPTION OF PLATES XVII AND XVIII.

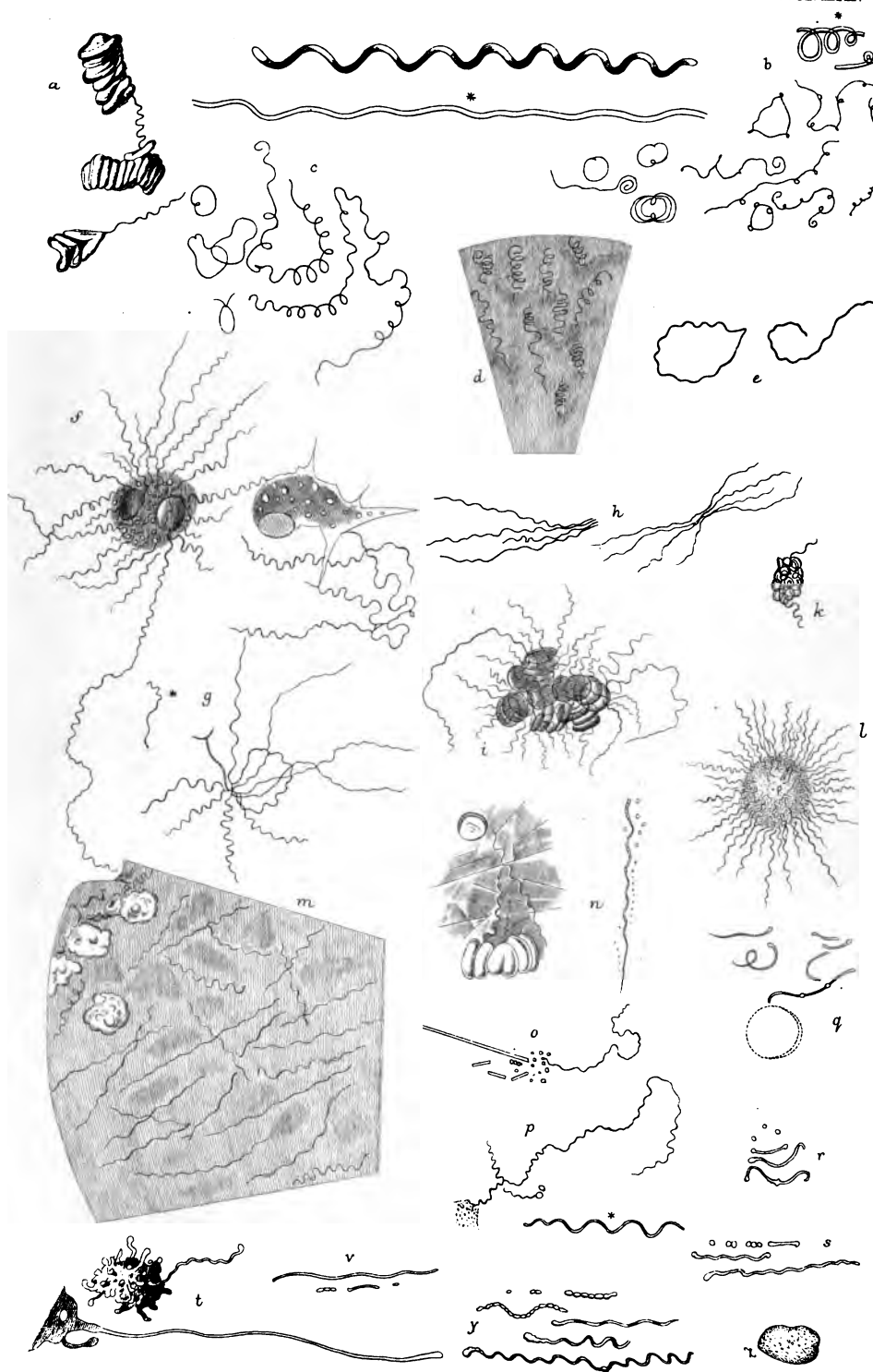
The Spirillum of Recurrent Fever. (Vandyke Carter, M.D.)

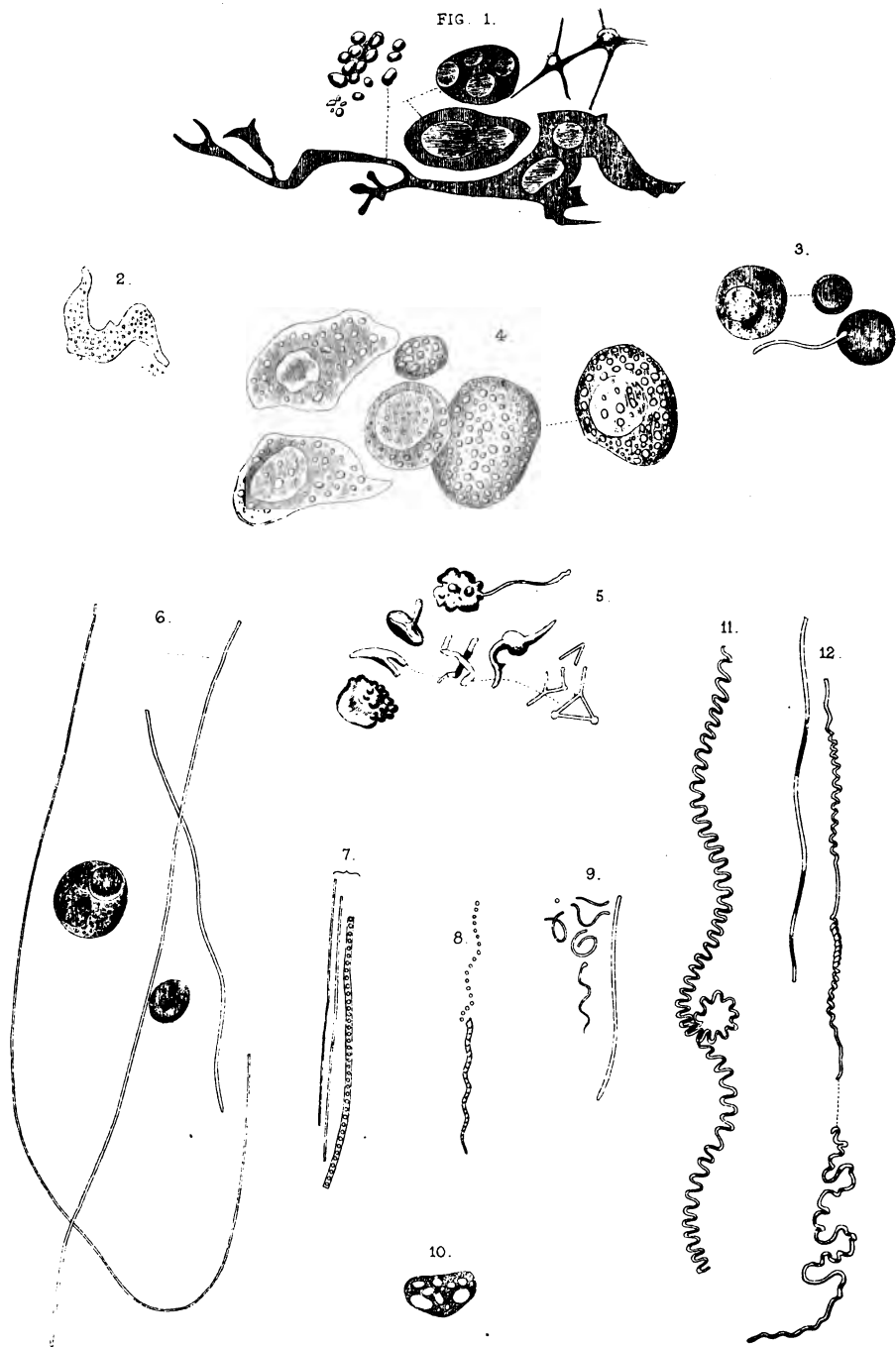
PLATE XVII.

- FIG. *. Spirillum in motion.
a—n. Spirillum of the blood.
o, p. Spirillum of saliva.
g, r, s. Filaments seen in ague, &c., in the blood.
t—s. Protoplasm endothelium filaments in the blood during life.

PLATE XVIII.

- FIGS. 1—5. Protoplasm endothelium filaments in the blood during life.
6—10. Rods, &c., in blood after death.
11, 12. Spirochæte bacillus from water of tanks.





|

|

A CASE
OF
INTUSSUSCEPTION,
IN WHICH
ABDOMINAL SECTION WAS PERFORMED.

BY
C. HANDFIELD JONES, M.B. CANTAB., F.R.S.,
PHYSICIAN TO ST. MARY'S HOSPITAL;

AND
HERBERT W. PAGE, M.C. CANTAB., F.R.C.S.,
ASSISTANT SURGEON TO ST. MARY'S HOSPITAL.

Received March 26th—Read May 28th, 1878.

THE following history may be deemed of sufficient interest to be brought before the Royal Medical and Chirurgical Society, as a continuation of the series of similar cases recorded in vols. lvii and lix.

R. S—, a boy, æt. five years, was admitted into St. Mary's Hospital, under Dr. Handfield Jones, on Dec. 31st, 1877. His mother states he has been ill six weeks, and that one doctor affirmed he had pleurisy, another typhoid. The child has lost flesh very much, and looks wan and weak. He suffers from severe paroxysms of pain in the abdomen lasting some minutes, and occurring every few

hours; in the intervals he seems quite free and is cheerful. On January 7th the abdomen was extensively dull, the bowels were loose, and the stools light coloured. Temperature normal. He was ordered—

R Ferri Carb. Sacchar., gr. v. Ter die.

Cod-liver oil. Port wine, 1 oz. Simple diet, milk and rice pudding.

9th.—Attacks of pain in the abdomen occur very irregularly, sometimes there are five or six a day, on other days scarcely any; their duration is very short. Abdomen resonant now. He seems to be improving, takes his food pretty well, and likes his wine.

14th.—Has been suffering much pain all night and this morning; was cheerful yesterday. He had much pain on the night of the 10th, and on the 11th. The pain is relieved by pressure. Abdomen is dull in left side at the depending part, the remaining two thirds are resonant. Ordered—

R Tinct. Opii, $\mathfrak{m}\mathfrak{i}\mathfrak{v}$, in enemate, dolore invadente.

And,

R Quiniae Sulph., gr. j;
Acid. Sulph. dil., $\mathfrak{m}\mathfrak{i}\mathfrak{j}$;
Sp. Chloroformi, $\mathfrak{m}\mathfrak{i}\mathfrak{j}$;
Aqua, $\mathfrak{z}\mathfrak{i}\mathfrak{j}$. Quater die.

To omit the Ferri Carbon.

17th.—Bowels open twice yesterday, once during the night; more pain occurs after food. Omit the quinine and take—

R Hydrarg. c. Cretâ, gr. $\mathfrak{i}\mathfrak{i}\mathfrak{j}$;
Pulv. Rhei, gr. j. Omni nocte.

19th.—Was much better all yesterday, and passed a good night, but is now (2 p.m.) complaining again of his belly, and does not care for food; the whole abdomen is more or less dull, except the stomach region, and a firm mass is felt in the left iliac fossa. He was quite bright and cheerful yesterday. Ordered—

R Tinct. Cinchon. flav., $\mathfrak{m}\mathfrak{x}\mathfrak{i}\mathfrak{j}$;
Sp. Ammon. c., $\mathfrak{m}\mathfrak{v}\mathfrak{j}$;
Aqua, $\mathfrak{z}\mathfrak{i}\mathfrak{j}$. Quater die.

21st.—In the evening and night of 19th he had much sickness and pain in abdomen; the tumour in left iliac fossa became very marked; there was no stool. The following day there was much collapse with pain and vomiting. I directed inflation to be performed per anum, which was done efficiently by Mr. Prowse, the house surgeon, the child's body being at the same time inverted. I saw him soon after the operation, he was lying in bed quiet, with a warm hand and a pulse of 128, and the collapse had passed off; to-day he is doing well and has no pain or sickness. Something resembling a cordy band is felt in or near the left iliac fossa.

22nd.—Pain came on again this afternoon, lasted four hours, and was not lessened by an opiate enema. Abdomen is dull, but not distended. Pulse distinct, 120. Tongue natural, no sickness. Ordered—

Adde Potass. Iodid., gr. j, ad ʒij, mist.

24th.—Brows knit; some pain to day, none yesterday. Abdomen resonant all over.

28th.—Aspect cachectic; does not take food well; has slight recurrences of pain.

31st.—Bowels quite regular. Has daily slight attacks of pain. Left side of abdomen notably duller than right. Temperature normal.

Feb. 4th.—The tumour has reappeared in the left iliac fossa, accompanied by pain, sickness, and bloody mucous stools. Inflation was again performed as before, and the tumour completely disappeared. Before inflation was used the child was inverted, his legs being laid across another person's shoulders, and his body well shaken, but without any change in the abdomen. Pulse very feeble.

R. Tinct. Opii, ℥iv;
Sp. Chloroformi, ℥v;
Aquæ, ad ʒij.

7th.—Is doing well; has no pain.

11th.—The left iliac tumour reappeared yesterday, and

was again removed by insufflation. Is complaining of pain to-day. Ordered—

R. Tinct. Nucis Vom., ℥iij;
Aq. Chloroformi, ʒj. Ter die.

14th.—The tumour reappeared in the usual situation on the 12th, and was attended by pain, but not by sickness; it disappeared without inflation. He is still very thin, but eats well.

18th.—Tumour recurred on the 16th, and was removed by inflation a fourth time. Abdomen is dullish on both sides.

21st.—Has pain in abdomen to-day, and has passed some slightly blood-tinged, flaky fluid.

25th.—Was in much pain this morning. A large tumour was both felt and seen in left iliac fossa, but no bloody mucus was passed. Insufflation was performed for the fifth time, and the tumour again disappeared.

27th.—Is merry this afternoon. His father took him out the next day, though warned that recurrence of the disorder was almost certain. He was readmitted at 9.30 a.m. on March 4th in an alarming condition, the symptoms having returned two days before. He was suffering much pain which he endeavoured to relieve by stooping with his head low, and was very pallid. A large tumour was felt in the left side of the abdomen, extending from the iliac to the umbilical region. The right side was tympanitic, the left dull. At 3.30 p.m. the lower bowel was again inflated, for the sixth time, but not with such satisfactory results as on the former occasions. The tumour became softer, but instead of disappearing only moved higher up. Almost as soon as the influence of the anæsthetic had passed off he was completely doubled up with pain. In the evening the inflation was again performed, and failing, water was injected in large amount, but with no better success. He had great pain, was constantly sick, and passed a quantity of mucus per anum mixed with blood, but no fæces.

March 5th.—No sleep all last night. He looks now very

pale and miserable, is in great pain, vomits immediately after taking anything, and passes much mucus per rectum. Pulse and heart's impulse scarcely perceptible. Tumour extends into epigastrium; right side of abdomen tympanitic. At five o'clock the operation described below was commenced, and was completed at 6.15 p.m. At 6.30 p.m. he was breathing distinctly and regularly, and swallowed the brandy and water given to him well.

The operation, with remarks, by MR. PAGE.

Having learnt from Dr. Handfield Jones that the symptoms had throughout been those of intussusception, and that insufflation, previously successful, had on the last occasion completely failed, I satisfied myself of the presence of a distinct hard lump in the left flank, but made no minute examination, as the child was cold and collapsed, and pulseless at the wrist.

The steam spray and other requisites of antiseptic treatment having been at once prepared, I began the operation at five o'clock in the ward, opening the abdomen in the median line below the umbilicus by an incision rather more than two inches long. It was at once noticeable that there was no general peritonitis. Inserting two fingers I explored the tumour, and felt it to extend from the left iliac region upwards in the course of the colon, and follow that of the transverse part until, about the middle line, it seemed to end deeply towards the posterior part of the umbilical region of the cavity. It was thus obviously of large size, and was so hard as almost to convey the impression of being some new growth connected with one of the viscera. Unable after careful search through this small opening to define the precise commencement of the invagination, I carried the incision upwards to a distance of half an inch above the navel. With the greater room thus given the upper limit of the tumour was distinctly found, and, as before said, was deeply placed. Effort was at once made by

gentle traction, and fixing of the tumour, to drag out the ensheathed part, but without avail. The small intestines were therefore withdrawn from the abdomen, and during their exposure they were either being played on by the antiseptic spray, or were covered with lint soaked in a warm solution of carbolic acid. The small intestines out of the way, it was easier to reach the volvulus, and I was able by simply pulling to extricate about two inches of the ensheathing part, which was found a good deal congested. Failing to get more by traction, my attention was next directed to the lower end of the mass, for I had in mind the experience of Mr. Hutchinson that pulling down the ensheathing part had effected the reduction, which pulling out the ensheathed part could not procure. By neither method could I make any further way, and deeming it necessary to examine more closely the large, hard, and resistant mass, I after some difficulty withdrew it also from the abdomen. It then became clear that the ensheathing part was entirely composed of large intestine, into which ileum was seen to pass. It was much distended, somewhat dark and congested at the upper extremity, but its surface was smooth and glistening, and there was no inflammatory lymph. Attempts at reduction being still unsuccessful, it was found on nearer inspection that the mass of colon was itself composed of an ensheathing and an ensheathed part; that there was, in fact, a second intussusception. In this, however, the order of things was reversed, for the lower part was included in the upper, which thus became the ensheathing portion; the whole forming a sausage-shaped body, the size of the child's ankle, from ten to twelve inches long. Passing a finger along each sheath it could be felt that the extremities of the two intussusceptions overlapped each other about the centre of the mass; and it became evident that this lower invagination must be released before the upper could be withdrawn. Grasping hold, therefore, of each half of this lower intussusception I exerted steady traction upwards and downwards, and slowly and with difficulty drew out some two inches of the ensheathed large

bowel, until such gentle efforts ceased to have avail. More power was evidently necessary. The parts began further to separate, and pulling in opposite directions almost as hard as I could, I gradually drew the lower out of the upper, and simultaneously the upper off the lower part of the invaginated colon. The ultimate point extracted was somewhat rough, and for about an inch slight adhesions were undoubtedly present; but at no point did the consistence or colour of this part of the gut appear markedly altered, so soon as the reduction was complete, and the natural calibre thereby restored. At this moment the state of the child seemed even more immediately alarming than before. It was thought to be dying, but it rallied after brandy had been given by the mouth.

I now returned to the higher or more ordinary intussusception. Dragging on the ensheathed part was without further influence, and I found that by simple traction at the lower end I was unable to draw off, or to start the drawing off of the ensheathing part. I therefore took the far end of the volvulus in my hand, and gently squeezing it, the two portions began at once to glide from one another, the ensheathed part being, as it were, pushed out of the hand in the act of closure, while the ensheathing was retained in the grasp. A repetition of this simple and delicate manœuvre completed the reduction, the vermiform appendix being the last to appear.

Close to the origin of the appendix there was some thickening and congestion of the cæcum. This part of the intestine was lying quite away from its normal site, being above the umbilicus and only just to the right of the middle line. There was not much difficulty in returning the intestines into the abdomen. All fluids poured into the cavity having been taken up with sponges, the parts were accurately brought together by silver sutures passed either through the peritoneum, or through the skin. The operation lasted an hour and a quarter, and during the latter part of it, the steam spray having come to an end, the hand spray was called into service. This was not very well kept up, and

only when the hands of the operator began to feel the numbing effects, was it known that a much stronger solution of carbolic acid, one in twenty, had with it been unfortunately used. To this the intestines were, for a short time exposed. The child was put to bed and hot bottles were applied. It rallied markedly during the evening, took food, spoke, had an action of the bowels and was once sick. When seen at eleven o'clock it was quite warm, and the radial pulse was distinctly perceptible, but the child was restless and seemed to be in pain. It sank exhausted on the following morning at 4 a. m., nine hours and three quarters after the operation.

Mr. Duncan, who made the *post-mortem* examination, reports as follows: "Abdomen only examined. The lips of the wound were found to be adherent to one another, but the adhesions were easily separable. To the anterior abdominal wall at the site of operation part of the great omentum and a few coils of small intestine were adherent. The coils of small intestine were more or less generally adherent to one another, though all the adhesions, above named, were very slight and easily separable. The affected portions of intestines were sticky, slightly injected, and covered with a thin layer of lymph on their free surfaces. Four and a half inches above the ileo-cæcal valve, a portion of the ileum measuring about two inches in length appeared greatly congested. On opening the bowel a circular anæmic stripe extended around the walls, measuring about half an inch, and being at its lower border about three inches above the valve. Above this sharply defined stripe, the intestine for two and a half inches was paler than the succeeding portion which was greatly congested, the mucous membrane being here swollen and the vessels much injected. The cæcum was of a blackish green hue, and its mucous membrane, especially that of the vermiform appendix, was much congested. The meso-cæcum was very lax; the cæcum 'floating,' as it were, and it could be easily drawn over the left iliac fossa. There appeared to be no peritonitis of the large intestine, and no affection of the mesenteric glands."

The several features of the case have been thus recorded in detail, for it is essential that the steps of an operation such as abdominal section, which has the best grounds for establishing itself as a justifiable procedure in surgery, should be told without reserve. The method whereby alone it seemed possible to unfold the upper or more usual intussusception fully confirms the experience of Mr. Hutchinson, recorded in the fifty-ninth volume of the 'Transactions of the Society.' Mr. Hutchinson found that the impediment to reduction was "clearly due to the ensheathing bowel being thrown into folds by traction, and thus constituting a series of strictures which gripped its contents." An endeavour thereupon to prevent this drawing into folds by holding the ensheathing layer straight revealed to him "the true method of reduction, for by pulling this downwards, instead of trying to pull the involved part upwards, he accomplished the replacement with the greatest ease." The remembrance of this was a great help to me. It was, in fact, evident that no amount of traction, at any rate, not so much as it was thought prudent to exert, had any influence in moving the distal end of the invagination. The actual mode of reduction had, indeed, been foreshadowed by Mr. Hutchinson, who, judging from his experience of the case which he records in the paper already named, had an impression that in future operations the "reduction ought to be accomplished by squeezing" the lower end of the invaginated tract.

The existence of a second intussusception, in which the usual order of ensheathing and ensheathed parts was reversed, a condition which Mr. Pollock (Holmes' 'System of Surgery') speaks of as "quite exceptional, even if it ever occur," formed an unforeseen complication in the case. Had it not been there, there seems no reason why the upper invagination should not have been at once reduced with ease; and its presence, moreover, necessitated that which should, as much as possible, be avoided—any long exposure of the intestines outside the abdominal cavity. The great distension of this part of the bowel, and the slight adhesions between the opposed surfaces at the farthest point of the involution,

CASE OF INTUSSUSCEPTION.

made the reduction of this intussusception a work of considerable difficulty; and although that was accomplished, as the *post mortem* proved, without damage to the structure of the gut, such forcible traction as was used might itself have been a source of peril.

Viewed from the serous surface at the time of operation, part of the implicated bowel was in a condition past repair, but the state of the mucous membrane, as described by Mr. Duncan, shows how imperative it is that, when other means have been tried and have failed, there should be no delay in surgical interference. The laxity of the mesocæcum is of interest as throwing light on a possible origin, and predisposing cause, of the intussusception.

Concluding remarks by Dr. HANDFIELD JONES.

The long continuance of attacks of pain in this case before the appearance of tumour or other positive sign of intussusception is very noteworthy. Taking into account the time he had been ailing before admission, these preliminary disorders extended over about eight weeks. The child's cachectic appearance, coupled with the abdominal pain, led me to imagine that some coarse lesion, such as tuberculosis affecting the intestines and peritoneum especially, was present. This, however, was not the case, and we seem to have no choice but to ascribe the pain and subsequent motor derangement to a functional disorder of the abdominal nerves and ganglia. As to the nature of this disorder we can only remark that, inasmuch as a relaxed state of a lower segment must certainly favour the intrusion of an actively contracting part above, it is most probable that the essential condition of intussusception is a local paralysis of the portion of the intestine which becomes the sheath of the volvulus. Neuralgic pain in other parts is often associated with motor paralysis, and probably is so in the abdominal organs. The frequent recurrences of intussusception were evidently dependent on the persistence of the morbid condition of the intestinal innervation.

The next point for notice is the existence of an ascending intussusception, the lower part of the sigmoid flexure of the descending colon being intruded into the transverse portion. There were, therefore, two volvuli coexisting and proceeding in opposite directions. The possibility of such an occurrence has been questioned by no less an authority than the late Dr. Brinton, who says that the existence of a single case of backward intussusception rests on no better evidence than that which establishes the reality of the sea-serpent, and such like marvels.

The third point is the absence of peritoneal inflammation and adhesions, after so many occurrences of intussusception. It was not until the sixth recurrence that inflation failed, and there is no reason to doubt that the procedure would have been equally successful on this occasion as on the preceding, had not the removal of the child from the hospital interposed a considerable delay to its performance, during which adhesions had time to form. This puts in a strong light the necessity of having early recourse to inflation, as soon as symptoms indicative of intussusception are present.



*Appendix to Dr. Harley's second communication on
Simple Atrophic Sclerema.*

(See page 101.)

September 11th.—William W— came to the hospital quite recovered from his attack, looking more cheerful and expressing himself as being better than usual; but on the evening of the same day, shortly after returning home, the sickness and diarrhoea recurred, and was followed by fatal collapse; the voice was reduced to a whisper, the extremities were cold and insensible and the hands purple. He died on the evening of the 13th and retained his clear intellect to the last.

I examined the body thirty-six hours after death. It was extremely emaciated, there had been no *rigor mortis*, and the muscles were still flaccid. The sclerema and stiffness of the wrists and finger-joints had completely passed off and there was no extraordinary lividity of the now soft and emaciated fingers. There was no trace of œdema anywhere, the skin was thin and supple, and almost every trace of fat had disappeared.

The liver, kidneys, suprarenal bodies, spleen, mesenteric glands, and pancreas were perfectly healthy. The gall bladder was contracted, and contained only a teaspoonful or two of recently secreted bile. The alimentary canal was healthy throughout, the stomach was thin and dilated, and contained, as did the intestines, a considerable quantity of watery fluid.

The costal cartilages were free from ossific deposit. The pericardium was thickened and inseparably adherent to the anterior surface of the right ventricle for the space of about

$\frac{1}{2}$ superficial inch. The heart was enlarged, weighing fourteen ounces. The right and left ventricles presented a striking contrast, the former being thin, dilated and flaccid, while the latter was hard and contracted, the wall being fully one inch thick, and the muscle very firm and healthy. The endocardium and valves were also healthy. A firm colourless clot extended a short way into the pulmonary artery, but excepting a minute soft clot or two, the blood was fluid, and in the left cavities also of a venous colour. Excepting the diaphragmatic surfaces, the lungs were everywhere adherent, and so firmly adherent that only a portion of the antero-lateral surface of the right could be separated without laceration; their tissue was crepitant and healthy.

The united pleuræ were thickened above and behind by cicatrix-like bands of tendinous strength and hardness. It was impossible to make a clean dissection of the surfaces even with the scalpel, and only small shreds could be torn away by the exertion of great force. The sympathetic cord of both sides, from the inferior cervical ganglion downwards, was so involved in the thickening that with the time and means at my disposal (the patient died at home) I could not make a satisfactory isolation. The blood-vessels and articular surfaces were healthy.

While the dead body thus exhibits plainly enough the cause of the so-called sclerema, it proves conclusively the insignificance, not to say the incorrectness, of this term. True there was hardening of the connective tissue of a portion of the body continuously for a period of seven years, and in some parts a consequent degeneration; but it is equally true that this hardening was merely the consequence of pressure of the fluids of the body on vessels which had lost their contractile power. The condition of the hands was for the whole of this period that of impending dry gangrene. In ordinary dry gangrene the paralysis of the vessels is due to local disease affecting the arterial walls; in the cases of sclerema above narrated it is due, as I have previously surmised, to an affection of the central ganglia.

It is an interesting fact that the condition above described

as simple atrophic sclerema may be one of the results of general pleurisy—a rare result doubtless, for I have never found adhesions so general and firm in any previous case; and a remote one also, due to progressive increase of the thickening, for the history shows that W. W— could not have had pleurisy since his attack of fever about seventeen years before his death.

The immunity of the lower extremities is now most satisfactorily explained. The loss of muscular tone in the stomach points to implication of the vagus, the gastric branches of which could scarcely have escaped. The simple hypertrophy of the heart was commensurate with the arterial palsy.

11

INDEX.

These Indices to the annual volumes are made on the same principles as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.

ABDOMINAL SECTION in a case of intussusception (C. H. Jones and H. W. Page) 301

AMNESIA, on a case of, with post-mortem examination (W. H. Broadbent) 147

Reference to the writer's paper on the "Mechanism of Speech and Thought," in vol. lv, 147; perceptive centres, 148; case in which their fibres of communication with the "naming centre" were destroyed by disease, 149; case of lesion of the name or idea centre itself, 149; post-mortem examination of ditto, 153-5; question of affixing a name to a disease in which the faculty of understanding words spoken by himself or others is lost by the patient, 156; results of lesion of similar parts of right and left hemisphere, 157-8.

ANEURISM, LARGE, of the aorta, innominate, subclavian, and carotid arteries, treated successfully by double distal ligation (R. Barwell) 13

Previous history, 13; present state, 14; tape and compass measurement of the tumour, 16; operation and after treatment, 17-24; temperature diagram, 20; remarks on the nature of the aneurism, operation, diet, &c., 24-7; result and deduction, 27; sequel and post-mortem, 28-32.

AORTA, see *Aneurism*.

ARTERIES, see *Aneurism*.

ARTERY, EXTERNAL CAROTID, ligature of, in treatment of hæmorrhage from punctured wounds of the throat and neck (W. H. Cripps) 229

BAKER, W. Morrant.

On the removal by operation of a hairy mole occupying half the forehead 33

INDEX.

Richard.

Large aneurism of the aorta, innominate, subclavian, carotid arteries, treated successfully by double distal anastomosis 13

see *Calculi*.

Change in the proportion of red corpuscles in, in some skin diseases (G. Thin) 95

Number of red corpuscles in blood in healthy persons, 95; in persons suffering from exhausting diseases, 96; M. Malassez's observations (note), 96; *Table* of numbers of red corpuscles in cases of skin disease, 98-9.

— moving bodies observed in, during life, in a case of noma (A. E. Sansom) 1

DIES, MOVING, observed in the blood during life in a case of noma (A. E. Sansom) 1

EMBRYO, see *Spirillum* Fever.

EXTERNAL BRANCHIAL FISTULÆ, cases of, in the external ears (Sir James Paget) 41

Reference to the first papers on the subject by Dzondi and Ascherson, and to Heusinger's collection of cases, 41-2; the case, 43-5; supernumerary auricles, 46-7; association of malformations of the external ear with harelip and cleft palate, 47; appendix of Mr. Cumberbatch's case of branchial fissure on the helix, 49.

BROADBENT, Wm. Henry, M.D.

On a case of amnesia, with post-mortem examination . 147

BUTLIN, Henry Trentham.

The microscopic anatomy of the smooth tongue, "chronic superficial glossitis" 51

CALCULI, account of 500 cases of operation for stone in the bladder of the male adult (Sir H. Thompson) . 159

All cases below twenty years of age and all female cases excluded, 160; notice of previous records—Cheselden, Martineau, Brodie, Fergusson, 160-1; analysis of the author's cases, 161; nature of the calculi, 163; relative proportions of lithotrity and lithotomy, 163; tables of the mortality following the two operations, 164-6; relative mortality of the cases of the different operators, 167-8; accidents met with in lithotrity: fracture of lithotrite and impaction of uric acid calculus between two blades, 169-70; accidents in lithotomy: perforation of bladder by staff, secondary hæmorrhage and wounding the rectum, 170-72; analysis of causes of death in the sixty-one cases in the tables, 173-4; after-history of elderly lithotrity patients, 175; danger of cystitis from prolonged series of sittings, 175; Mr. Clover's apparatus for removing fragments by aspiration, 176; use of the lithotrite for removal of phosphatic deposits, 177; relative proportions of cases fitted for either operation, 177-8.

- CARTER, H. Vandyke, M.D.**
Notes on the spirillum fever of Bombay, 1877 . . . 273
- CASTS**, see *Tubular Casts*.
- COATS, Joseph, M.D.**
On the pathology of tetanus and hydrophobia . . . 79
- CORPUSCLES, RED**, see *Blood*.
- CRETINOID AFFECTION** in middle-aged women, the condition of "myxœdema" in (W. M. Ord) . . . 57
- CRIPPS, William Harrison.**
Treatment of hæmorrhage from punctured wounds of the throat and neck, especially considered with regard to ligature of the external carotid artery . . . 229
- EARS**, cases of branchial fistulæ in the external ears (Sir James Paget) . . . 41
- EYE**, internal muscles of, paralysis of, probably indicating disease of the lenticular ganglion (Jon. Hutchinson) . . . 215
Anatomy of the lenticular ganglion, 216; symptoms of its destruction: paralysis of the circulating and radiating fibres of the iris and of the ciliary muscle, 216; seven cases illustrative of this group of symptoms, 217-22; general summary, 222-7; definitions of terms used: paralytic mydriasis and myosis, iridoplegia, cycloplegia, and ophthalmoplegia interna, 224-5; table of cases, 228.
- FEVER**, see *Spirillum Fever*.
- FISTULÆ**, see *Branchial Fistulæ*.
- FOREHEAD**, on the removal by operation of a hairy mole occupying half the (W. M. Baker) . . . 33
- GANGLION, LENTICULAR**, disease of, indicated by paralysis of the internal muscles of the eye (Jon. Hutchinson) . . . 215
- GLOSSITIS**, chronic superficial, microscopic anatomy of (H. T. Butlin) . . . 51
— see *Tongue* (smooth).
- HÆMOPHILIA**, contribution to the pathology of (Percy Kidd) . . . 243
History of the case, 243; post-mortem examination, 245; extensive proliferation of the endothelial cells lining the vessels, 247-50.
- HÆMORRHAGE** from punctured wounds of the throat and neck, especially considered with regard to ligature of the external carotid artery (W. H. Cripps) . . . 229
Circumstances of emergency in which difficulties arise, 229; analysis of the death ratio in ligature of the common carotid artery,

230-1; brain symptoms, 231; recurrence of hæmorrhage from original wound, 232; objections to the operation, 234; table showing rarity of wounds of the internal carotid, 235; reasons in favour of the operation, 236; table of cases in which the common carotid was tied for a wound in the throat or neck, 237.

HÆMORRHAGE, PULMONARY, on the pathological traces of
(Reg. E. Thompson) 253

Relation of pulmonary hæmorrhage to pulmonary phthisis, 253; opinions of Hoffman, Niemeyer, Bäumler, &c., as to traces of previous hæmorrhage found in lungs after death, and pneumonic products, 255; true pathology of these nodules, preferential regions they inhabit, microscopical appearances, 255-8; necrosis of the nodules, either by gradual erosion or general liquefaction, 258-60; mortar-like patches, their form and apparent origin, 260; conclusions, 261; appendix of cases, 262-70.

HAIRY MOLE, see Mole.

HARLEY, John, M.D.

Second communication on simple atrophic sclerema . . 101

— appendix: sequel of case of Wm. M., post mortem,
&c. 313

HUTCHINSON, Jonathan.

On paralysis of the internal muscles of the eye (ophthalmoplegia interna), a group of symptoms which probably indicates disease of the lenticular ganglion . . 215

HYDROPHOBIA and TETANUS, on the pathology of (Jos. Coats) 79

INTUSSUSCEPTION, case of, in which abdominal section was performed (C. H. Jones and H. W. Page) . . 301

Clinical history of the case, 301-5; the operation, with remarks by Mr. Page, 305; post-mortem examination by Mr. Duncan, 308; concluding remarks by Dr. H. Jones, 310.

JONES, C. Handfield, M.B., and Herbert W. PAGE, M.C.

Case of intussusception, in which abdominal section was performed 301

KIDD, Percy, B.A.

Contribution to the pathology of hæmophilia . . 243

KIDNEY, on some points in the minute anatomy of, and their relation to the pathological phenomenon of tubular casts (Reg. Southey) 201

Discovery of the ascending collecting, and down-looping tubes by Henle, 201; opinion of the author in opposition to Henle, that they form a continuous system, 202; mode of commencement and course of the tubuli uriniferi, 202-5; renal casts, pathological solution of the mode of their formation, 206-10; resistance of fatty degeneration by the down-looping tubes, 210; formation of casts in urine of colloid substance from the excretory system of urine tubes, 211; other varieties of casts, 212.

LIGATURE, see *Artery* (external carotid).

— double distal, for large aneurism of the aorta, innominate, subclavian, and carotid arteries (R. Barwell) . . . 13

LITHOTOMY, *table* of deaths following, in Sir H. Thompson's practice 165

— see *Calculi*.

LITHOTRITY, *table* of deaths following, in Sir H. Thompson's practice 164

— see *Calculi*.

LUNGS, see *Hæmorrhage* (pulmonary).

MOLE, HAIRY, occupying half the forehead, on the removal of, by operation (W. M. Baker) 33

MUSCLES, INTERNAL, of the eye, paralysis of, probably indicating disease of the lenticular ganglion (Jon. Hutchinson) 215

MYXŒDEMA, an essential condition in the cretinoid affection occasionally observed in middle-aged women (W. M. Ord) 57

Reference to Sir W. Gull's paper on the cretinoid state of women in adult life, 57; Case 1 described, 57-61; post-mortem notes, 61; Case 2 described: marked symptoms, slowness of thought, articulation, and muscular action, 63-4; jelly-like state of the fibrillar or white element of the connective tissue, a common condition of these cases, 66; other pathological conditions, 66-8; analogy of the state of the skin with sclerema, 68; result of varnishing the skin of animals, 68; condition of the hands in this disease, and of those of cretins, 69-70; the symptoms, the effects of jelly-like swelling of the connective tissue padding of peripheric termination of sensory nerves, 71; application of these effects to the origin of cretinism, 72; antagonism between goitre and sporadic cretinism, 73; report on mucin from the skin, by Dr. C. Charles, 74; report of post-mortem examination, by Dr. W. S. Greenfield, 75.

NOMA, on a case of, in which moving bodies were observed in the blood during life (A. E. Sansom) 1

General history of the case, 2; chart of temperature, pulse, and respiration, 4; autopsy, 4; examination of the blood during life, 5; inoculation experiments upon animals, 8.

OPHTHALMOPLÉGIA INTERNA, a group of symptoms probably indicating disease of the lenticular ganglion (Jon. Hutchinson) 215

ORD, *William Miller*, M.D.

On myxœdema, a term proposed to be applied to an essential condition in the cretinoid affection occasionally observed in middle-aged women 57

